EVALUATION OF CLINICAL PROFILE OF RENAL FAILURE – A STUDY OF 153 PATIENTS

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CERTIFICATE

This is to certify that the dissertation titled "**Evaluation of Clinical Profile of Renal Failure - A Study of 153 Patients**" is the bonafide original work of **DR. S. N. MEENAKSHI SUNDARI** in partial fulfillment of the requirements for **M.D. Branch - I (General Medicine)** Examination of the Tamilnadu DR. M.G.R Medical University to be held in March 2007. The period of study was from May 2005 to July 2006.

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DECLARATION

I, **Dr. S. N. MEENAKSHI SUNDARI,** solemnly declare that dissertation titled **"Evaluation of Clinical Profile of Renal Failure - A Study of 153 Patients"** is a bonafide work done by me at Government Stanley Medical College and Hospital during May 2005 and July 2006 under the guidance and supervision of my unit chief **Prof. S. SHIVAKUMAR**, M.D., Professor of Therapeutics.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I)** in **General Medicine.**

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TABLE OF CONTENTS

INTRODUCTION

Renal failure is an important medical complication for which patients are admitted in medical wards. Renal failure is classified as Acute Renal Failure (ARF) and Chronic Kidney Disease (CKD). ARF is a reversible renal disease, while CKD is an irreversible renal disease.

On admission, it may be difficult to diagnose ARF or CRF and further evaluation is required to confirm the diagnosis. In our Hospital, these patients were admitted in the Medical Units, Nephrology Department or the Intensive Medical Care Unit. In this study patients admitted and diagnosed to have renal failure in medical units only were analysed. Acute diarrheal disease is the most common cause of ARF. Chronic glomerulonephritis is the most common cause of CKD which was closely followed by Diabetic Nephropathy. The study was done in Stanley Medical college Hospital, which is located in North Chennai. This study deals with etiological, clinical and laboratory profile of renal failure.

AIM OF THE STUDY

1. To evaluate the type of renal failure (ARF/CKD) admitted in the medical units.
2. To analyse the etiology, clinical features and management of ARF.
3. To analyse the etilogy, clinical feature and management of CKD.

ACUTE RENAL FAILURE

ARF is a syndrome characterized by rapid decline of glomerular filtrarion rate (hours to days), retention of nitrogenous waste products and pertubation of extracellular fluid volume and electrolyes and acid base homeostasis1.ARF complicates 2- 5% of hospital admission and 30% of ICU admissions1,2. Most ARF are reversible.

The manifestations of ARF are subtle. Losing the function of one half of the nephron mass(1 million glomeruli) will cause creatinine levels to rise from about 0.7 mg/dL upto only about 1.4 mg/dL[2]. Oliguria (urine output < 400ml/d) is the commonest clinical feature (50%). ARF is usually asymptomatic and diagnosed when biochemical monitoring of hospitalized patients shows an increase in blood urea and creatinine concentration. The ADQI (Acute Dialysis Quality Initiative) evolved the RIFLE classification, which grades ARF according to severity (Risk, Injury, Failure) and outcome (Loss and End stage disease).

The RIFLE system

The term Acute Kidney Injury if preferred to Acute renal failure[3]. Plasma creatinine and or urine volume indicate kidney function. A change in baseline S. creatinine reflects the severity of ARF.

- **RISK** - S.creatinine increases 1.5 times or GFR decreases by >25% and fall of GFR occurs in 6 hours

- **INJURY**- S.creatinine increases 2 times or GFR decreases by >50% and fall of GFR occurs in 12 hours
- **FAILURE**- S.creatinine increases 3 times or GFR decreases by >75% and fall of GFR occurs in 24 hours or anuria for > 12 hours or S creatinine > 4 mg/dL
- **LOSS**- persistent ARF, complete loss of kidney function for >4 weeks
- **ESKD**- End stage kidney disease for > 3 months

ARF is divided into three categories:

1. PRERENAL ARF (50%) - Diseases that cause renal hypoperfusion without affecting renal parenchyma
2. INTRINSIC RENAL ARF (40%) - Diseases that affect renal parenchyma.
3. POSTRENAL ARF (5%)- Diseases causing urinary tract obstruction

PRERENAL ARF

It is the most common form of ARF. It is a physiologic response to renal hypoperfusion due to a decrease in effective arterial blood volume. Decreased effective circulating volume may result from volume depletion, peripheral vasodilatation or low cardiac output. Prerenal ARF is rapidly reversible upon restoration of renal blood flow and glomerular ultrfiltration pressure. There is no damage to renal parenchyma.

ETIOLOGY OF PRERENAL ARF

1. HYPOVOLUMIA
    a) hemorrhage, burns, dehydration
    b) gastrointestinal fluid loss- vomiting, diarrhea, surgical drainage
    c) sequestration of fluid in extravascular space- pancreatitis, peritonitis
2. LOW CARDIAC OUTPUT

    Diseases of myocardium, valves, pericardium, cardiac arrythmias, tamponade.

3. ALTERED RENAL SYSTEMIC VASCULAR RESISTANCE RATIO
    a) Systemic vasodilatation- sepsis, anaphylaxis
    b) Renal vasoconstriction- hypercalcemia, epinephrine, norepinephrine, cyclosporine, tacrolimus. Amphotericin B
    c) Cirrhosis with ascites- hepatorenal syndrome
4. HYPERVISCOSITY SYNDROMES

    Multiple myeloma, macroglobinemia, polycythemia.

PATHOGENESIS OF PRERENAL ARF

Autoregulation occurs maximally at mean systemic arterial B.P of 80 mm Hg. Further fall in B.P leads to decline in GFR leading to ARF. Drugs affecting the compensatory mechanisms may connvert compensated renal hypoperfusion to Prerenal ARF or trigger progression of Prerenal ARF to Ischemic intrinsic renal ARF. These drugs cause inhibition of prostglandin synthesis like NSAIDS, ACEI and ARB. NSAIDS precipitate prerenal ARF in patients with hypovolumia.

In patients with bilateral renal artery stenosis or unilateral stenosis with single kidney, the GFR is solely dependant on Angiotensin II . Therefore Angiotensin II blockers will precipitate ARF in these patients.

In prerenal states the kidney avidly retains sodium, usually resulting in low urine sodium and fractional excretion of sodium (FE_{Na}) of less than 1%. Other conditions where FE_{Na} >1% may be observed are radiocontrast induced renal failure, AGN, liver failure, pigment induced nephrotoxicity, early obstructive nephropathy, vasculitis and normal renal function.

FE_{Na} is calculated as follows:

$FE_{Na} = [(U_{Na} X P_{cr})/(P_{Na} X U_{cr})] X 100$

Where U= Urine and P= Plasma[4].

ETIOPATHOGENESIS OF INTRINSIC RENAL FAILURE

1. **Acute tubular necosis**
   a) ischemic- etiology same as prerenal ARF(hypovolumia, low cardiac output, systemic vasodilation) or by obstetric complications (abruptio placentae, postpartum hemorrhage)
   b) Toxins

Endogenous toxins- rhabdomyolysis, hemolysis, uric acid, oxalates

Exogenous toxins – radiocontrast, cyclosporine, antibiotics

2. **Diseases of the Glomeruli or renal vasculitis.**
   a) glomerulonephritis- PIGN, MPGN, RPGN and vascilitides

b) hemolytic uremic syndrome, TTP, DIC, Toxemia of pregnancy, Accelerated hypertension, radiation nephritis, SLE, Scleroderma

3. **Interstitial Nephritis**
    a) Allergic- Antibiotics (β lactums, sulphonamides, trimethoprim, rifampicin), NSAIDS, Diuretics, captopril, cimentidine, thiazides
    b) Infections- acute pyelonephritis, leptospirsis, candidiasis
    c) Lymphoma, leukemia, sarcoidosis.
4. **Renovascular obstruction**
    a) Renal Artery obstruction- atherosclerotic plaques, thrombosis, embolism, vasculitis.
    b) Renal vein obstruction- thrombosis, compression.
5. **Intratubular deposition and obstruction**- myeloma protein, uric acid, oxalate, acyclovir, Methotrexate, sulphonamides
6. **Renal allograft rejection.**

ACUTE TUBULAR NECROSIS (ATN)

ATN is the most common form of intinsic ARF. Prerenal azotemia is the soil that nurtures ATN. In ischemic ARF, hypoperfusion induces ischemic injury to renal parenchymal cells, particularly tubular epithelium and recovery takes 1 to 2 weeks after normalisation of renal perfusion as it requires repair and regeneration of renal cells[5].

ATN has four phases.

1. Initiation phase (1-12 hours) - This begins when renal blood flow decreases resulting in cellular ATP depletion which trigger pathways to sub lethat cell injury or cell death.
2. Extension phase (10- 60 hours) - this is characterised by
   a) continued hypoxia of the outer nmedulla due to intra renal vasoconstriction by Angiotensin II, Endothelin-1, decreased NO
   b) stasis and accumulation of RBC's and WBC's in the outer medullary vessels
   c) endothelial damage leading to continued ischemia
   d) promotion of coagulation in peritubular vessels
   e) inflammatory injury due to activation of inflammatory cascade

   Renal injury can be limited by restoration of blood flow during this period.

3. Maintenance phase - Renal blood flow returns to near normal. The tubular cells undergo apoptosis, proliferation, dedifferentiation, migration and re-establish strctural integrity. GFR stabilises and stays low (5-10 mL/min) because of intra renal vasoconstriction, urine output is lowest and uremic complications arise.
4. Recovery phase - characterized by repair and regeneration of tubule epithelial cell and gradual return of GFR to near normal. This phase may be complicated by a marked diuretic phase.

NEPHROTOXIC ARF

The incidence of Nephrotoxic ARF is increased in elderly, in patients with chronic renal insufficiency. Intrarenal vasoconstriction is the main event in radiocontrast nephropathy. Volume depletion, multiple myeloma, heart failure and age >65 years are risk factors. The ARF is oliguric and S.creatinine peaks in the first 72 hours after exposure. Renal functions recover in 7-10 days.

Aminoglycoside nephrotoxicity occurs in 10-30% of the courses of aminoglycosides, even in therapeutic levels. It is most often nonoliguric and is caused by direct toxicity to the tubule epithelial cells[4]. The other drugs are acyclovir,foscarnet, amphotericin B, pentmidine and chemotherapeutic agents like cisplatin, carboplatin and ifosfamide.

The most common endogenous toxins are calcium, myoglobin, hemoglobin, urate, oxalate and myeloma light chains. Hypercalcemia causes intrarenal vasoconstriction and calcium deposition within the kidney. Myoglobinuria with ARF complicates 30% of cases of Rhabdomyolysis like traumatic crush injury, seizures, excessice exercise, heat stroke, alcohol intoxication, cocaine intoxication and infectious or metabolic disorders.

It is associated with muscle pain, increased uric acid, hyperkalemia, hyperphosphatemia and increased level of creatine kinase(MM isoenzyme). ARF due to hemolysis follows massive blood transfusion reactions. They cause ARF by direct toxicity to tubule epithelial cells, inducing intratubular cast formation,

causing intrarenal vasoconstriction by inhibiting nitric oxide bioactivity. Both hemolysis and rhabdomyolysis induce ARF especially in acidotic and hypovolemic individuals.

In ARF with multiple myeloma, intratubular casts containing immunoglobulin light chains and other proteins are formed and cause myeloma cast nephropathy. Light chains are also directly toxic to the tubule epithelial cells. Acute uric acid nephropathy complicates treatment of lymphoproliferative are myeloproliferative disorders causing intratubular precipitation of massive amounts of uric acid.

Certain drugs induce ARF by triggering allergic interstitial nephritis. There is infiltration of the tubulointerstitium by granulocytes (mostly eosinophils), macrophages and lymphocytes. AIN presents as sudden onset of azotemia in association with signs of generalized drug reaction or with septicemia or malignant infiltration of the kidney. Interstitial edema, intense patchy or diffuse cellular infiltration and relatively well-preserved glomeruli and tubules characterize renal histology. Eosinophilic infiltration occurs early but disappears rapidly. The chance of finding eosinophiluria is dependent upon the timing of urinalysis[6].

Patients with atherosclerosis can develop ARF due to embolization of cholesterol crystals to the renal vasculature. They also have retinal arteriolar plaques, lower extremity livedo reticularis and necrosis in distal digits[4].

Acute GN can result in ARF. RPGN presents with an acute deterioration of renal function, nephrotic or non-nephrotic proteinuria and active urinary sediment with hematuria and RBC casts. Oliguria may be present. RPGN can be further characterized by the presence of immune complex deposition (SLE, poststreptococcal GN, IgA nephropathy, endocarditis), the paucity of immune complex deposition (Wegener's granulomatosis, microscopic polyangitis, Churg-Strauss syndrome) or the presence of anti-GBM disease.

MALARIAL ARF

The renal failure sets within 5-7 days after the onset of fever. The most important histopathological abnormality was ATN. The factors contributing to tulular injury are hypovolemia, intravascular hemolysis, intravascular coagulation, septicemia, catecholamine release and haemo-rheological changes induced by parasitized erythrocytes[7,8].

LEPTOSPIRAL ARF

The pathogenesis of renal failure is leptospirosis is multifactorial and may include hypoxia secondary to hypovolemia/hypotension and direct nephrotoxicity due to toxic by-products of leptospires[9]. Body fluid loss due to vomiting,

increased insensible water losses and diminished intake of fluid are responsible for hypovolemia and hypotension in some cases. In the kidney, leptospires initially cause glomerular injury and by hematogenous spread, the organisms

reach peritubular capillaries and migrate to the interstitium, renal tubules and tubular lumen causing interstitial nephritis and tubular necrosis[10].

POST RENAL ARF

This accounts for less than 5% of the case of ARF. ARF from Obstruction requires obstruction to urine flow between the external urethral meatus and bladder neck, bilateral ureteric obstruction or unilateral ureteric obstruction in a patient with single functioning kidney. The presence of alternating anuria and polyuria is classic occurence in urinary tract obstruction. Urinary tract obstructions may be within the urinary tract (eg. Blood clots, stones, sloughed papillae or fungal balls) or extrinsic (eg tumors, retroperitoneal fibrosis or even inadvertent ligation). Bladder neck obstruction is the most common cause of post-renal ARF and is commonly due to prostatic diseases, neurogenic bladder or treatment with anticholinergics. During the early stages of obstruction, continued glomerular filtration leads to increased intraluminal pressure upstream to the site of obstruction. As a result there is gradual distention of the proximal ureter, renal pelvis, calyces and a fall in GFR..

CLINICAL FEATURES OF ARF

Prerenal ARF has symtoms such as thirst and dizziness and physical evidence of orthostatic hypotension and tachycardia, reduced JVP, decreased

skin turgor, and dry mucus membranes. History of recent initiation of treatment with NSAIDS, ACE inhibitors or angiotensin II blockers should be sought.

Intrinsic renal ARF follows severe renal hypoperfusion complicating hypovolemic or septic shock or following major surgery.

Postrenal ARF presents with suprapubic and flank pain due to distention of the bladder and renal collecting system. Colicky flank pain radiating to the groin suggests acute ureteric obstruction. Neurogenic bladder should be suspected in patients receiving anticholinergics or with physical evidence of autonomic dysfunction.

Complications of ARF include expansion of ECF volume with pulmonary edema, hyperkalemia, hyperphosphatemia, metabolic acidosis, anemia and uremic syndrome.

INVESTIGATIONS IN ARF

URINALYSIS

Anuria suggests complete urinary tract obstruction or severe intrinsic or prerenal ARF. P^H is acidic in prerenal ARF and alkaline in obstructive uropathy. Glucosuria without hyperglycemia suggests severe proximal convoluted tubule damage.

Hyaline casts are found in prerenal ARF. Hyaline casts are formed from Tamm-Horsfall protein, which is secreted by epithelial cells of the loop of Henle.

Pigmented “muddy brown” granular casts are characteristics of ATN. There will be associated microscopic hematuria and mild tubular proteinuria. RBC casts indicate glomerular injury. White cell casts and nonpigmented granular casts suggest interstitial nephritis. Eosinophiluria (> 5% urine leukocytes) occurs in antibiotic-induced allergic interstitial nephritis. Eosinophiluria also occurs in atheroembolic ARF.

Minimal proteinuria occurs in prerenal and obstructive disorders. Glomerulonephritis causes selective albuminuria whereas ATN, AIN and post-renal ARF cause nonselective proteinuria. Light chain proteinuria as in multiple myeloma causes neagative distick test for protein but gives a positive flocculation test. Positive reaction for blood occurs in hemoglobinuria and myoglobinuria. If the urine is heme positive but the serum is not pink red, myoglobinuria is suggested. Red pink plasma suggests hemoglobinuria[6].

LABORATORY FINDINGS

Hyperuricemia, hyperkalemia, hyperphosphatemia with increased levels of LDH indcates acute urate nephropathy and tumor lysis syndrome. A wide serum anion and osmolal gap indicates ethylene glycol or methanol ingestion. Systemic eosinophilia occurs in allergic interstitial nephritis, atheroembolic disease and polyangitis nodosa. Ultrasound is useful to exclude postrenal ARF. Retrograde or antegrade pyelography helps to identify the site of obstruction. Doppler ultrasound and magnetic resonance angiography are useful to assess

the patency of renal arteries and veins. Renal biopsy is reserved for patients in whom pre renal and post renal ARF have been excluded and the cause of intrinsic renal failure is unclear.

MANAGEMENT OF ARF

FLUID MANAGEMENT

Fluid replacement should be equal to insensible losses plus urinary and other drainage losses. Hyponatemia in patients with ARF is usually secondary to volume expansion with hypotonic fluid, whereas hypernatremia is caused by overly aggressive diuresis. Fluid challenge can be given in oliguric patietns who are not volume overloaded. Around 500-1000 ml of normal saline is infused over 30-60 min.

DIETARY MODIFICATION

Total calorie intake should be 35-50 kcal/kg/d. Salt intake should be restricted to 2-4 g/d. Potassium intake should be restricted to 40 mEq/d and Phosphorus intake should be restricted to 800 mg/d.

Patients at risk for radiocontrast nephropathy should be well hydrated with 75-150 ml/hour of 0.45% saline beginning 12-24 hours before the contrast study and ending 12 hours after the study. Acetylcysteine (600 mg PO bid; 4 doses total, starting 1 day before the procedure) reduces the incidence and severity of contrast nephropathy[11].

In rhabdomyolysis aggressive fluid administration should be initiated to replace fluid that is lost into the necrotic muscle and to establish high urine flow. Alkalinization of the urine(urine P^H >6.5) by iv infusion of 2-3 ampoules of $NaHCO_3$ in 1 L of 5% dextrose in water increases the solubility of heme pigments and will hasten recovery.

In acute uric acid nephropathy, uric acid production is decreased by administration of allopurinol 600 mg PO before cytotoxic therapy followed by 100-300 mg/day. Forced alkaline diuresis helps to prevent uric acid precipitation[4].

A one-week course of prednisone 60 mg PO once daily will hasten the recovery in drug induced acute interstitial nephritis. Acute glomerulonephritis or vasculitis may respond to glucocorticoids, alkylating agents and plasmapheresis.

Control of systemic hypertension is essential in malignant hypertensive nephrosclerosis, toxemia of pregnancy and other vascular diseases. Antihypertensives that do not decrease renal blood flow (clonidine, prazosin, calcium channel blockers) are used for controlling hypertension.

Leptospirosis is treated with Penicillin G 1.5 million units 6^{th} hourly and Doxycycline 100 mg twice daily[9]. Malaria is treated with Chloroquine 25 mg/kg or quinine 10 mg/kg 8^{th} hourly.

Obstruction of urethra or bladder neck is usually managed by transurethral or suprapubic placement of a bladder catheter. Ureteric obstruction is initially treated by percutaneous catheterization of the dilated renal pelvis or ureter.

Obstructing lesions may be removed percutaneously (calculus, sloughed papilla) or bypassed by insertion of a ureteric stent (carcinoma).

Hyperkalemia is treated calcium gluconate, insulin, glucose, bicarbonate, potassium binding resins. Hyperkalemia that is resistant to medical therapy is an indication for urgent dialysis. Metabolic acidosis is treated with sodium bicarbonate.

DIALYSIS

Hemodialysis and Peritoneal dialysis appear equally effective in the management of ARF. The dialysis modality is chosen according to the needs of individual patients. Peritoneal dialysis is preferable if the patient is hemodynamically unstable. The indications for dialysis are uremic syndrome, refractory hypervolemia, hyperkalemia and acidosis.

PROGNOSIS

Mortality rates vary according to the cause of ARF. It is around 60% following trauma or major surgery, 30% in toxin related ARF and 15% in obstetric patients. 50% of the patients who survive an episode of ARF have subclinical impairment of renal function. 5% of patients never recover function and require long term renal replacement with dialysis or transplantation[1].

COMPARISON OF ETIOLOGICAL DATA

The following studies were done by the Department of Nephrology, Madras medical college, during 1979-84, 1987-91, 1995-2004 and the change in the etiological profile of ARF in South India were analysed[12-14].

ETIOLOGY	1979-84 %	1987-91 %	1995-2004 %
Medical causes		90	87.6
Leptospirosis	5.3	31	7.5
Acute diarrheal disease	23.5	30.5	28.6
AGN	26.2	8.5	9.3
Snake bite	3.2	4.7	7.8
Drugs	5.3	5.4	13.4
Copper sulphate poisoning	11.2	3.4	4.3
Obstetric causes	8.5	9	8.9
Surgical causes		1.5	3.4

In all the three studies, acute diarrheal disease remained the most common cause of ARF. The incidence of Leptospiral ARF increased to 31% during 1987-91, but has decreased now to 7.5%. Drugs have become the second most common cause of ARF, which was mostly due to excessive use of over-the-counter analgesics. Sepsis and malaria have become the emerging causes. Despite improvements in antenatal care, obstetric renal failure remains a significant cause of ARF. The incidence of Surgical ARF was on the rise. The mortality was 19.6% n the recent study, but was 27.8% in the first study and 26.4% in the second study.

CHRONIC KIDNEY DISEASE

Chronic kidney disease is a pathophysiologic process lasting for more than 3 months, with multiple etiologies, resulting in the inexorable attrition of nephron number and function and frequently leading to end stage renal disease (ESRD)[15].

Kidney damage is either functional or structural with or without reduction in GFR as identified by any one or all of three criteria

1. Abnormal gross or microscopic pathology
2. Laboratory markers of kidney damage in blood, urine, imaging tests
3. GFR < 60 ml/min/1.73 m^2 in the absence of any other renal abnormality[16]

Risk factors for CKD include family history of renal disease, hypertension, diabetes, autoimmune diseases, older age, past episode of ARF, Systemic infections, Urinary tract infections, Urinary stones, Lower urinary tract obstruction, Drugs including NSAID abuses[16].

STAGES OF CKD

A recently widely accepted international classification divides CKD into a number of stages defined by clinical estimation of GFR[15].

STAGE	DESCRIPTION	GFR, mL/min/1.73m^2
1	At increased risk, kidney damage with normal or increased GFR	90(with CKD risk factors)
2	Kidney damage with mildly decreased GFR	60-89
3	Moderately decreased GFR	30-59
4	Severely decreased GFR	15-29
5	Renal failure	<15

During stages 1 and 2, patients remain symptom free. As the decline in GFR progresses to stages 3 and 4, clinical and laboratory compliations of CKD become prominent. Patients may be azotemic and erythropoietin production decreases. Abnormalities of calcium and phosphorus metabolism causes metabolic bone disease and abnormalities of sodium, water, potassium and acid-base homeostasis occurs. When the GFR falls to <15 ml/min/1.73 m^2, uremia occurs wherein continued survival is dependant upon renal replacement therapy (dialysis or transplantation).

ESTIMATION OF GFR

GFR cannot be measured directly. The most widely used measures of GFR are based on the 24 hrs creatinine clearance or serum creatinine concentration. GFR can be estimated by one of the two equations shown below:

a) Cockcroft-Gault Equation

$$\text{Estimated Creatinine clearance (mL/min)} = \frac{(140 - \text{Age}) \times \text{Body weight (kg)}}{72 \times P_{cr}\ (\text{mg/dL})}$$

b) Modification of diet in renal disease study (MDRD)

$$\text{Estimated GFR (mL/min/1.73 m}^2) = 1.86 \times (P_{cr})^{-1.154} \times (\text{age})^{-0.203}$$

ETIOLOGY

Diabetic and hypertensive nephropathy are the leading causes of CKD and ESRD. Hypertension is a common cause and consequence of CKD in the elderly. Other causes include Nondiabetic glomerular disease (nephritic or nephrotic), cystic kidney disease and tubulointerstitial diseases.

The clinical features of CKD are shown in this table[17].

SYSTEM	CLINICAL MANIFESTATIONS
Electrolytes	Edema, hyponatremia, hyperkalemia, metabolic acidosis, hyperuricemia, hyperphosphatemia, hypocalcemia
Gastrointestinal	Anorexia, nausea, vomiting, malnutrition
Cardiovascular	Accelerated atherosclerosis, systemic hypertension, pericarditis
Hematologic	Anemia, immune dysfunction, platelet dysfunction
Musculoskeletal	Renal osteodystrophy, muscle weakness, growth retardation in children, amyloid arthropathy
Neurologic	Encephalopathy, seizures, peripheral neuropathy
Endocrine	Hyperlipidemia, glucose intolerance caused by insulin resistance, amenorrhea and infertility in women, impotence
Skin	Pruritus

Pathophysiology of uremia

The pathogenesis of uremia involves three major mechanisms. They are diminished excretion of electrolytes and water, reduced excretion of organic solutes, and decreased hormone production[18,19] .

Diminished excretion of electrolytes and water

An important function of the healthy kidney is to excrete the electrolytes and water generated from dietary intake in order to maintain a steady state in which intake and urinary excretion are roughly equal. The number of functioning nephrons at the stage of ESRD is so small that urinary excretion cannot achieve a level equal to intake. Clinical manifestations include edema and hypertension (caused by sodium retention), hyponatremia (resulting from free water retention), hyperkalemia, metabolic acidosis, hyperuricemia, and hyperphosphatemia.

Reduced excretion of organic solutes

The kidneys excrete a variety of organic solutes, the commonly measured ones being urea and creatinine. The excretion of urea and creatinine is not actively regulated and the plasma level of these solutes begin to rise with the decline in GFR..

Decreased hormone production

The kidneys normally produce several hormones, including erythropoietin and calcitriol (1,25-dihydroxycholecalciferol), the active form of vitamin D. The decreased production of these two hormones plays an important role in the development of anemia and bone disease respectively.

Systemic complications of CKD and their treatment

Uremic syndrome consists of an array of complex symptoms and signs that occur when advanced kidney failure prompts the malfunction of virtually every

organ system. Patients may have symptoms such as malaise, weakness, insomnia and a general feeling of being unwell. Patients may lose their appetite and complain of morning nausea and vomiting.

Electrolyte disturbances

Sodium balance: Sodium balance remains virtually normal until very late in the course of CKD, because the kidney can markedly increase the amount of sodium excreted per nephron by reducing tubular sodium reabsorption. Intake of large amounts of sodium can easily overwhelm the excretory capacity of the failing kidney and result in fluid retention, edema, and hypertension. Likewise, if diuretics are used overzealously, the patient may become volume-depleted. Clinically evident edema is uncommon until the GFR falls to less than 15 mL/min/1.73m^2. Edema also occurs in patients with glomerular disease and significant proteinuria (nephrotic syndrome) and in those with heart failure. The cornerstone of treatment of edema (and hypertension) is restriction of dietary sodium to a level lower than that recommended for uncomplicated hypertension (<100 mEq/day; 2.3 g of sodium or 6 g of salt).

Thiazide diuretics are usually ineffective if the serum creatinine level is greater than 3 mg/dL. Loop diuretics are the agents of choice in patients with CKD. Patients with advanced CKD may require doses of furosemide (Lasix) as high as 400 mg per day. Lack of response to high doses of loop diuretics often is due to noncompliance with dietary sodium restriction.

Potassium-sparing diuretics (spironolactone) are contraindicated because of the risk of inducing hyperkalemia.

Potassium balance: Potassium balance and plasma potassium level are also maintained until very late in CKD, mainly because of an increase in renal excretion of potassium per functioning nephron and an increase in potassium output in the stool[20]. Hyperkalemia may occur in association with dietary indiscretion (eg, excessive consumption of chocolate, dried fruits, or bananas), use of potassium-containing salt substitutes, increased catabolism (as with severe intercurrent illness), or metabolic acidosis. It may also be seen with the use of potassium-sparing diuretics, angiotensin-converting enzyme (ACE) inhibitors, and nonsteroidal anti-inflammatory drugs (NSAIDs). Patients with a serum potassium concentration below 6 mEq/L usually respond to a combination of a loop diuretic and a low-potassium diet. Asymptomatic patients with a serum potassium level above 6 to 6.5 mEq/L can be treated with sodium polystyrene sulfonate, given orally or by colonic enema. Symptomatic hyperkalemia, particularly in the presence of electrocardiographic changes, is treated with combinations of intravenous calcium gluconate and infusions of glucose and insulin with or without bicarbonate. This therapy transiently drives potassium into the cells. In patients with kidney failure, dialysis may be required.

Water balance: The ability to concentrate or dilute urine is impaired in patients with CKD, which makes them more susceptible to hypernatremia and

hyponatremia. Hypernatremia may occur if water consumption is not sufficient to replace fluid loss. More commonly, hyponatremia develops in patients with CKD because they either drink water or are given hypotonic fluids in excess of their ability to excrete water.

Metabolic acidosis: Most patients with CKD develop metabolic acidosis because of their reduced ability to excrete the hydrogen ions. The goal is to maintain the serum bicarbonate level above 20 mEq/L. Sodium bicarbonate is recommended. Calcium carbonate, which is often used as a phosphate binder, can help control acidemia as well.

Cardiovascular complications

The most common cause of death in patients with ESRD is cardiovascular disease. Increased prevalence of coronary artery disease in CKD is both due to traditional risk factors like hypertension, hypervolemia, dyslipidemia, sympathetic overactivity and hyperhomocysteinemia and non traditional risk factors like anemia, hyperphosphatemia and a state of microinflammation that occurs in CKD. The elevated acute phase reactants like interleukin 6 and C-reactive protein contribute to the coronary occlusive process[15]. Myocardial ischemia and left ventricular hypertrophy along with salt and water retention in uremia lead to congestive cardiac failure.

Hypertension almost invariably develops in patients with CKD and is usually volume-dependent. The goal is to achieve a blood pressure of less than

130/85 mm Hg. Treatment of hypertension includes restriction of dietary sodium and use of diuretics. ACE inhibitors are protective in patients with proteinuria .

Uremic pericarditis was once a common finding but is seen much less often today because dialysis is usually started before it appears. Treatment of this condition includes intensive dialysis and the use of NSAIDS[21].

Hematologic complications

A normochromic and normocytic anemia develops in most patients when the GFR falls below 60 mL/min/1.73 m^2. It occurs mainly as a result of erythropoietin deficiency and deficiency in iron, folate, or vitamin B_{12}. Correction of anemia with erythropoietin results in improved cardiac function, exercise tolerance, central nervous system symptoms, appetite, and sexual function. Although white blood cell count is usually within normal range when CKD is present, the function of these cells may be defective, leading to an increased susceptibility to infections. Bleeding tendency occurs due to defective platelet function[22-23].

Bone disease

Metabolism of calcium and phosphorus is abnormal in patients with CKD and is associated with the development of bone disease. Phosphate retention occurs as GFR declines. Both hyperphosphatemia and reduction in the active form of vitamin D lead to hypocalcemia. As attempts are made to normalize the serum

calcium level, secondary hyperparathyroidism can develops and causes renal osteodystrophy, which includes osteitis fibrosa, osteomalacia and adynamic bone disease[24]. Calcium carbonate and calcium acetate are effective phosphate binders which also correct hypocalcemia. Vitamin D preparations like calcitriol help to suppress the levels of parathormone and correct hypocalcemia.

Neurologic complications

Cerebrovascular accidents of all types are common in CKD. Uremic encephalopathy is characterized by insomnia, impairment of concentration, alterations in usual sleep rhythms, emotional lability and depression. Dialysis produces rapid clearing of the mental state and correction of abnormal electroencephalographic findings. Generalized motor seizures may also occur in patients with advanced kidney failure. A symmetrical polyneuropathy of a mixed sensory-motor type also occurs.. Sensory symptoms present first (cramps, paresthesias, restless legs). Disturbances in autonomic function may cause postural hypotension and impotence[25].

Management of CKD

Dietary modification includes Protein restriction to 0.6-0.8 g/kg/d of high biologic value protein, Potassium restriction to 40 mEq/d, no added salt diet and salt restriction to 2-3 g/d, fluid restriction to output plus 500 ml.

Anemia is treated with recombinant human erythropoietin 50-100 U/kg SC two or three times a week. The management of complications are already discussed.

Renal replacement therapy

This is indicated for the patients with ESRD.

Hemodialysis: This works by diffusion of small molecular weight solutes across a semi permeable membrane. Fluid removal occurs via ultrafiltration. Dialysis is usually performed three times a week. Complications are nausea, vomiting, headache, hypotension, bleeding due to anticoagulation used in HD, dialysis associated pericarditis.

Peritoneal dialysis: PD can be used for ARF and ESRD. It uses peritoneum as dialysis membrane. Dialysis exchange is performed by infusion of 2 L fluid into the peritoneal cavity followed by equilibration period and dialysate drainage. Complications include peritonitis, hyperglycemia due to systemic absorption of glucose from PD fluid and protein loss.

Renal transplantation: This offers the patient a lifestyle closest to normal. Pretransplantation evaluation of the recipient is done which includes blood group compatibility testing and human lymphocyte antigen typing. The recipient needs life long immunosuppression to prevent rejection[4].

PATIENTS AND METHODS

1. Patients aged 13 and above, admitted to the medical units of Government Stanley Hospital with Serum Creatinine > 1.5 mg/dl were taken up for the study. They were further subdivided into:
   a. Acute Renal Failure (ARF)
   b. Chronic Kidney Disease (CKD)

2. The following criteria were used for selecting patients for ARF and CKD.
   a. ARF
      - Patients with Serum Creatinine $\geq$ 1.5 mg/dl in whom the renal failure was reversible with treatment
      - Rapid onset of renal failure
      - Normal sized Kidneys
   b. CKD
      - Patients with Serum Creatinine $\geq$ 1.5 mg/dl, whom the renal failure was not reversible with treatment
      - Gradual onset of renal failure

3. Exclusion Criteria

   Patients admitted in Intensive Medical Care Unit and Nephrology Department were not taken up for the study. Patients admitted in the IMCU had mostly hospital acquired ARF and it has a high mortality rate (40-70%). The nephrology department was a referral centre and patients there do not comprise the model of the local population in Chennai.

4. The following data were obtained.

   a. Symptoms

   - Fever
   - Myalgia
   - Headache
   - Diarrhea
   - Vomiting
   - Giddiness
   - Altered Sensorium
   - Dyspnea
   - Chest Pain
   - Oliguria
   - Dysuria
   - Hematuria
   - Pedal Edema
   - Facial Puffiness
   - Drug Intake

   b. Signs

   - Pulse Rate (PR)
   - Blood Pressure
   - Elevated Jugular Venous Pressure (JVP)
   - Anemia
   - Jaundice
   - Dehydration
   - Pulmonary Edema

   c. The past history of systemic hypertension (SHT), Diabetes Mellitus (DM), Diabetic Nephropathy (DN), Chronic Kidney Disease (CKD) & Coronary Artery Disease (CAD) and their duration were sought.

   d. The following Investigation were done

      1. Blood Sugar
      2. Renal function tests- Blood urea, Serum Creatinine

3. Serum Electrolytes
4. Complete Hemogram
5. Urinalysis- Urine albumin
6. Liver Function Tests
7. ECG
8. Chest X-Ray
9. QBC for Malarial Parasites
10. MSAT/MAT
11. Ultrasound Abdomen

e. The treatment strategies involving Conservative Medical Management and Renal Replacement Therapies (Peritoneal Dialysis, Hemodialysis) were evaluated.

    a. Conservative Management of ARF

        - Fluid restriction to < 1 L/d
        - Salt restriction 1-2 g/d
        - Protein 0.6 g/kg/d of high biologic value
        - Potassium free diet
        - Carbohydrate 100 g/d
        - Diuretics
        - Restoring hemodynamic compromise due to fluid loss with Intravenous fluids

- Specific treatment- Malaria- Chloroquine 25mg/day, C. Doxycycline 100 mg twice daily or iv Quinine 10 mg/kg 8th hourly, Leptospirosis- iv crystalline penicillin
- Withdrawal of nephrotoxic drugs
- Forced alkaline diuresis

b. Renal replacement therapies for ARF

- Hemodialysis till recovery
- Peritoneal dialysis

c. Conservative Management of CKD

- Fluid restriction to < 1 L/d
- Salt restriction 1-2 g/d
- Protein 0.6 g/kg/d of high biologic value
- Potassium free diet
- Calories 35 Kcal/kg/d
- Antihypertensives
- Tight glycemic control
- Iron, calcium and vitamin supplements

d. Renal replacement therapies for CKD

- Hemodialysis
- Peritoneal dialysis
- Renal transplant

RESULTS

153 Patients were taken up for the study.

- Total number of Patients: 153
- Number of ARF: 71 (46%)
- Number of CKD: 82 (54%)
- Number of Males: 102 (67%)
- Number of Females: 51 (33%)

The Sex distribution is shown in TABLE 1.

TABLE 1

SEX DISTRIBUTION OF RENAL FAILURE

DIAGNOSIS	MALE	FEMALE	TOTAL NUMBER (n-153)
ARF	54	17	71 (46%)
CKD	48	34	82 (54%)
TOTAL	102 (67%)	51 (33%)	153

TABLE 2

AGE AND SEX DISTRIBUTION IN ARF

AGE (YEARS)	MALE	FEMALE	TOTAL (n-71)
13-20	2	1	3 (4%)
21-30	6	-	6 (8%)
31-40	8	1	9 (13%)
41-50	8	6	14 (20%)
51-60	11	5	16 (23%)
>60	19	4	23 (32%)
TOTAL	54 (76%)	17 (24%)	71

- Mean Age: 52.5 YEARS
- Range: 14-88

75% of the patients were above the age of 41 years. 76% were males and

24% were females.

TABLE 3

ETIOLOGY OF ARF

S.NO.	ETIOLOGY	TOTAL (n-71)
1	ACUTE DIARRHEAL DISEASE	11 (16%)
2.	MALARIA	8 (11%)
3.	LEPTOSPIROSIS	8 (11%)
4.	CONGESTIVE CARDIAC FAILURE	8 (11%)
5.	DRUGS	6 (8%)
6.	SEPSIS	6 (8%)
7.	GASTROINTESTINAL BLEEDING	3 (4%)
8.	ACUTE GLOMERULONEPHRITIS	3 (4%)
9.	DIABETIC KETOACIDOSIS	2 (3%)
10.	OBSTRUCTIVE UROPATHY	2 (3%)
11.	ACUTE PYELONEPHRITIS	2 (3%)
12.	RHABDOMYOLYSIS	1 (1.4%)
13.	ACUTE PANCREATITIS	1 (1.4%)
14.	UNCLASSIFIED	10 (14%)

The most common cause of ARF is Acute diarrheal disease. Infective diseases contributed to 50% of the ARF.

TABLE 4
CLINICAL FEATURES OFARF

S. NO	SYMPTOMS	TOTAL (n-71)
1.	FEVER	44 (62%)
2.	VOMITING	33 (46%)
3.	GIDDINESS	24 (34%)
4.	DYSPNEA	22 (31%)
5.	OLIGURIA	20 (28%)
6.	MYALGIA	17 (24%)
7.	DIARRHEA	16 (22%)
8.	PEDAL EDEMA	16 (22%)
9.	ALTERED SENSORIUM	14 (20%)
10.	HEADACHE	11 (15%)
11.	FACIAL PUFFINESS	8 (11%)
12.	DYSURIA	8 (11%)
13.	SEIZURES	5 (7%)
14.	HEMATURIA	4 (6%)

TABLE 5

S.NO.	SIGNS	TOTAL (n-71)
1	ANEMIA	24 (34%)
2.	CRACKLES	14 (20%)
3.	RAISED JVP	12 (17%)
4.	ASCITES	7 (10%)
5.	HEMIPLEGIA	6 (8%)
6.	JAUNDICE	6 (8%)

Fever was the most commonest symptom and anemia was the most commonest sign.

TABLE 6 shows the causes of fever in the patients with ARF.

TABLE 6

ARF WITH FEVER

S.NO.	ETIOLOGY	TOTAL (n-44)
1	ACUTE DIARRHEAL DISEASE	9 (20%)
2.	MALARIA	8 (18%)
3.	LEPTOSPIROSIS	8 (18%)
4.	SEPSIS	5 (11%)
5.	ACUTE PYELONEPHRITIS	2 (5%)
6.	PYOPNEUMOTHORAX	1 (2%)
7.	PUO	11 (25%)

PUO were the most common cause of fever followed by ADD, Malaria and Leptospirosis.

TABLE 7
CLINICAL PROFILE OF THE COMMON CAUSES OF ARF

CLINICAL PROFILE	ADD (n-11)	MALARIA (n-8)	LEPTOSPIROSIS (n-8)	CCF (n-8)	DRUGS (n-6)	SEPSIS (n-6)
MALE	8	6	5	6	5	4
FEMALE	3	2	3	2	1	2
MEAN AGE	50	44	48	62	52	58
AGE RANGE	19-70	14-65	14-70	42-80	41-88	46-70
FEVER	9	8	8		2	5
MYALGIA		5	4			
DIARRHEA	11		3			
VOMITING	8	5	5			
HEADACHE		4	3			
DYSPNEA		3		6		2
OLIGURIA	5	5		3	3	4
ANEMIA		6	3			3
MEAN BLOOD UREA	71	92	75	75	74	78
MEAN S. Cr	2.4	4.2	2.6	2	3.5	3.3
RANGE OF S. Cr	1.8-4.8	1.7-17.1	1.5-4.0	1.6-3.1	1.9-7	1.8-5.4

TABLE 8

BLOOD PRESSURE IN ARF

BLOOD PRESSURE (mm Hg)	NUMBER (n-71)
<90/60	9 (13%)
PREHYPERTENSION 120-139/80-89	3 (4%)
STAGE I HYPERTENSION 140-159/90-99	7 (10%)
STAGE 2 HYPERTENSION >160/100	15 (21%)

- Mean: 125/80 mm Hg
- Range: 60-210/40-130 mm Hg

13% of the people had hypotension BP < 90/60 mm Hg

TABLE 9

ANEMIA IN ARF

HEMOGLOBIN (g%)	NUMBER (n-71)
10-8	28 (40%)
8-5	11 (15%)
<5	-

- Mean: 8.5 g%
- Range: 6-10 g%

TABLE 10

BLOOD UREA IN ARF

BLOOD UREA	NUMBER (n-71)
40-59	29 (41%)
60-79	11 (4%)
80-89	11 (4%)
≥100	16 (23%)

- Mean: 79 mg%
- Range: 4-288 mg%

TABLE 11

SERUM CREATININE IN ARF

SERUM CREATININE	NUMBER (n-71)
1.5-2.9	53 (75%)
3.0-4.9	12 (17%)
≥5	6 (8%)

- Mean: 2.7 mg%
- Range: 1.5- 17.1 mg%

75% of patients with ARF had S. Creatinine < 3.0 mg% which shows that majority had mild renal failure.

TABLE 12

SERUM SODIUM IN ARF

SERUM SODIUM	NUMBER (n-71)
<135	24 (34%)
135-145	40 (56%)
>145	7 (10%)

- Mean: 137 mEq/L
- Range: 118-152 mEq/L

34% of patients had hyponatremia.

TABLE 13

SERUM POTASSIUM IN ARF

SERUM POTASSIUM	NUMBER (n-71)
<3.5	13 (18%)
3.5-5	51 (72%)
>5	7 (10%)

- Mean: 4.1 mEq/L
- Range: 2.3-5.8 mEq/L

TABLE 14

URINE ALBUMIN IN ARF

URINE ALBUMIN	NUMBER (n-71)
Trace	17 (24%)
+	9 (12%)
++	4 (5%)
+++	4 (5%)
++++	1 (1%)

Significant proteinuria is present in 11% of patients.

TABLE 15

RENAL REPLACEMENT THERAPIES IN ARF

RENAL REPLACEMENT THERAPY	NUMBER (n-71)
PD	1(1%)
HD	2(3%)

- Mortality: 2 (3%).

CHRONIC KIDNEY DISEASE

Number of CKD: 82

TABLE 16 shows the age and sex distribution in CKD

TABLE 16

AGE AND SEX DISTRIBUTION IN CKD

AGE (YEARS)	MALE	FEMALE	TOTAL (n-82)
13-20	-	2	2 (2%)
21-30	1	1	2 (2%)
31-40	8	5	13 (16%)
41-50	13	4	17 (21%)
51-60	10	7	17 (21%)
>60	16	15	31 (38%)
TOTAL	48 (59%)	34 (41%)	82

- Mean Age: 55 years
- Range: 13-82 years

80% of the patients were above the age 40 years

TABLE 17

ETIOLOGY OF CKD

S. NO.	ETIOLOGY	NUMBER (n-82)
1.	CHRONIC GLOMERULNEPHRITIS (CGN)	40 (49%)
2.	DIABETIC NEPHROPATHY (DN)	37 (45%)
3.	OBSTRUCTIVE UROPATHY	6 (7%)

CGN was the most commonest cause of CKD which was closely followed by Diabetic Nephropathy.

TABLE –18 shows the comparative age and sex distribution between the two most common causes of CKD – CGN and DN.

TABLE 18

AGE AND SEX DISTRIBUTION OF CKD and DN

AGE (YEARS)	CGN (n-40)			DN (n-37)		
	MALE	FEMALE	TOTAI	MALE	FEMALE	TOTAI
13-20	-	2	2	-	-	-
21-30	1	1	2	-	-	-
31-40	5	2	7	3	3	6
41-50	9	1	10	3	3	6
51-60	2	1	3	5	6	11
>60	12	4	16	3	11	14
TOTAL	29 (73%)	11 (27%)	40	14 (38%)	23 (62%)	37
MEAN	48			56		
RANGE	13-82			34-78		

In CGN, the Mean age was 48, while in DN it was 56 years. In CGN there was male preponderance (73%), but in DN females (62%) were more.

CLINICAL FEATURES OF CKD

TABLE 19

S. NO.	SYMPTOMS	NUMBER (n-82)
1	PEDAL EDEMA	57 (70%)
2.	OLIGURIA	44 (54%)
3.	DYSPNEA	40 (49%)
4.	FACIAL PUFFINESS	34 (49%)
5.	GIDDINESS	29 (35%)
6.	VOMITING	24 (29%)
7.	CHEST PAIN	14 (17%)
8.	DYSURIA	8 (10%)
9.	HICCUPS	5 (6%)
10.	HEMATURIA	2 (2%)

TABLE 20

S. NO.	SIGNS	NUMBER (n-82)
1	ANEMIA	47 (57%)
2.	CRACKLES	25 (30%)
3.	ASCITES	17 (21%)
4.	RAISED JVP	15 (18%)
4.	ANASARCA	12 (15%)
5.	HEMIPLEGIA	6 (8%)

Leg swelling was the most common symptom and anemia was the most common sign.

TABLE 21 and 22 show the past history of DM, DN, SHT,CAD, CKD in all the patients with CGN and DN.

TABLE 21

PAST HISTORY IN CGN

NO. OF YEARS	CGN (n-40)		
	SHT	CAD	CKD
<1	5	-	5
1-5	7	4	4
>5	5	-	2
TOTAL	17 (43%)	4 (10%)	11 (28%)

- SHT: Systemic Hrpertension
- CAD: Coronary artery Disease
- DN: Diabetic Nephropathy

Past history of Systemic hypertension was present in 43% of patients with CGN. 28% of patients with CGN were known patients of CKD.

TABLE 22

PAST HISTORY IN DN

NO. OF YEARS	DN (n-37)				
	DM	DN	SHT	CAD	CKD
<1	1	4	4	1	2
1-5	13	11	10	2	2
>5	14	1	6	-	1
Total	28 (76%)	16 (43%)	20 (54%)	3 (8%)	5 (14%)

76% of patients with DN were known cases of DM. 54% of patients with DN had past history of SHT.

TABLE 23

BLOOD PRESSURE IN CKD

BLOOD PRESSURE (mm Hg)	NUMBER (n-82)
PREHYPERTENSION 120-139/ 80-89	5 (6%)
STAGE 1 HYPERTENSION 140-159/ 90-99	17 (21%)
STAGE 2 HYPERTENSION ≥160/ 100	42 (51%)
ACCELERATED HYPERTENSION >210/130	11 (13%)

- MEAN BP: 152/92 mm Hg
- RANGE: Systolic 90-220/ Diastolic 60-170 mm Hg

72% of CKD patients were hypertensive. Most of them had Stage 2 hypertension (≥160/100 mm Hg) which shows that control of hypertension is poor.

TABLE 24

ANEMIA IN CKD

HEMOGLOBIN (g%)	NUMBER (n-82)
10-8	24 (29%)
8-5	26 (32%)
<5	1 (1%)

- MEAN HEMOGLOBIN – 8.1 g%
- RANGE- 4-10 g%

TABLE 25

BLOOD UREA IN CKD

BLOOD UREA (mg%)	NUMBER (n-82)
40-59	13 (16%)
60-79	26 (32%)
80-89	14 (17%)
≥100	29 (32%)

- MEAN- 87 mg%
- RANGE- 40-231 mg%

TABLE 26

SERUM CREATININE IN CKD

SERUM CREATININE (mg%)	NUMBER (n-82
1.5-2.9	45 (55%)
3.0-4.9	18 (22%)
≥5	19 (23%)

- MEAN- 3.7 mg%
- RANGE- 1.5-13.2 mg%

23% of patients had severe renal failure with S.Creatinine of >5.0mg%

TABLE 27

SERUM SODIUM IN CKD

SERUM SODIUM (mEq/L)	Number (n-82)
<135	20 (24%)
135-145	47 (57%)
>145	15 (18%)

- MEAN- 139 mEq/L
- RANGE- 114-152 mEq/L

TABLE 28

SERUM POTASSIUM IN CKD

SERUM POTASSIUM (mEq/L)	NUMBER (n-82)
<3.5	15 (18%)
3.5-5	57 (70%)
>5	10 (12%)

- MEAN: 3.9 mEq/L
- RANGE: 2.2-6.7 mEq/L

12% of patients with CKD had hyperkalemia.

TABLE 29 compares the proteinuria between CGN and DN.

TABLE 29

URINE ALBUMIN IN CKD

URINE ALBUMIN	CGN(n-40)	DN(n-37)	TOTAL(n-82)
Trace	9(22%)	7(19%)	16(20%)
+	8(20%)	12(32%)	20(24%)
++	8(20%)	12(32%)	20(24%)
+++	1 (3%)	3 (8%)	4 (5%)
++++	1 (3%)	1 (3%)	2 (2%)

Significant proteinuria occurred in 26% of patients with CGN and 43% of patients with DN.

TABLE 30

ECG IN CKD

ECG	NUMBER (n-82)
LVH	31 (38%)
CAD/ISCHEMIA	13 (16%)
SINUS TACHYCARDIA	5 (6%)
TALL T WAVES	5 (6%)

38% of patients had evidence left ventricular hypertrophy in ECG.

TABLE 31

CHEST X-RAY IN CKD

CHEST X-RAY	NUMBER(n-82)
CARDIOMEGALY	43 (53%)
PLEURAL EFFUSION	12 (15%)
PULMONARY EDEMA	6 (7%)

53% of patients with CKD had cardiomegaly in Chest X-Ray. These

. patients also had ECG evidence of LVH

TABLE 32

USG KIDNEYS IN CKD

Size (cm)	Number (n-82)
9-8	15 (18%)
8-7	33 (40%)
<7	11 (13%)

40% of patients had kidney size in the range of 8-7 cm.

TABLE 33

RENAL REPLACEMENT THERAPIES

Renal Replacement Therapy	Number(n-82)
PD	5 (6%)
HD	6 (7%)

- MORTALITY: 1(1%)

DISCUSSION

Kidney diseases are increasing day by day. It is important that these diseases are recognised early and referred at the appropriate time. Renal failure can be ARF or CKD. The incidence of ARF is around 1% of all the patients admitted to the general hospitals[14]. The prevalence of CKD in India is 5 to 10 per one lakh of the population. The increasing prevalence of chronic kidney diseases (CKD) has now been recognised as a major public health problem globally. Of the one lakh ESRD patients emerging every year, only 9,000 are put on dialysis. Out of this, 60 per cent drop out and 20 per cent die due to inadequate dialysis.

In this study renal failure was diagnosed utilising S.creatinine ≥1.5 mg/dl as the criteria. It is difficult to diagnose ARF or CKD at the first evaluation. As ARF is a reversible renal disease and CKD is an irreversible renal disease, it is absolutely essential not to make an error in diagnosis. The diagnosis of ARF or CKD was arrived only after further evaluation and observation for reduction in S.creatinine. In addition, acute on chronic ARF can make diagnosis difficult. In this study, all patients were labelled as renal failure as diagnosis and further classified as ARF or CKD only after evaluation.

Our Study screened 153 patients with S.Creatinine ≥1.5 mg/dL. Of these, 71 (46%) patients had ARF and 82 (54%) patients had CKD. Of these 102 (67%) were males and 51(33%) were females. The mean age was 53 years.

ACUTE RENAL FAILURE

71 patients had ARF. The Age distribution for ARF varied from 14-88. The Mean Age was 52.5 years. Out of 71 patients, 54 (76%) were males and 17 (24%) were females.

ETIOLOGY

The most common cause of ARF was found to be acute diarrheal disease (16%). The other causes are malaria (11%), leptospirosis (11%), congestive cardiac failure (11%), drug induced ARF (8%), sepsis (8%), gastrointestinal bleeding(4%), Acute glomerulonephritis (4%), diabetic ketoacidosis (3%), obstructive uropathy (3%), acute pyelonephritis(3%). The other rare causes were rhabdomyolysis(1%), acute pancreatitis(1%). For 14% of patients the etiology was not surely defined and they were categorized as unclassified. Among the unclassified, most had pyrexia of unknown origin which might be malaria or Leptospirosis, but the investigations were negative. Infections contributed to around 50% of the ARF. Our study did not invlove the obstetric and surgical causes of ARF because we have included only the patients in the medical wards. The 2 patients with obstructive uropathy were admitted in the medical ward as renal failure and subsequently the etiology was defined by the means of investigations.

M. Jayakumar et al showed in a study done in chennai during 1995-2004, published in 2006 that the medical causes contributed to 88% of the ARF

followed by obstetric causes (9%) and surgical causes (3%). Among the medical causes acute diarrheal disease (28%) remained the common cause of ARF, followed by drugs (13%), glomerulonephritis (9%), sepsis (9%), snake bite (8%), leptospirosis (8%), malaria (4%), Copper Sulphate (4%). Our study did not include the patients in the intensive medical care unit and therefore data of snake bite and Copper Sulphate poisoning who get admitted directly in the IMCU, were not there in our study[14].

RP Mathur and A Bhargav showed that in a study done in 2003-2004, the predominant etiological factors were septicemia (22%), alcoholic liver disease (19%), acute gastroenteritis (15%), falciparum malaria (8%), cardiac failure (7%), cirrhosis (7%), pneumonia (5%), stroke (5%) and others (13%) Others includued causes like benign prostatic hypertrophy with obstructive uropathy, post operative ARF, leptospirosis and trauma[26].

Sharma VK and Dubey TN reported that hypovolemia is the commonest cause of ARF. The etiological causes in their study were acute gastroenteritis with hypovolemia 28%, falciparum malaria 23%, obstrutive uropathy 13%, pregnancy related 8%, septicemia 8%, hepatorenal 5%, snake bite 4%, AGN 3%, NSAID induced 3% and miscellaneous 5%[27].

CLINICAL FEATURES OF ARF

The most common symptoms in our study were fever 62%, vomiting 46%, giddiness 34%, dyspnea 31%, oliguria 28%, pedal edema 22%. The other symptoms diarrhea 22%, altered sensorium 20%, headache 15%, dysuria 11%,

hematuria, seizures. Fever being the most commonest symptom implicates that infective diseases were the commonest cause of ARF.

RP Mathur and A Bhargav reported that the most common presenting symptoms were fever 46%, vomiting 35%, jaundice 27%, diarrhea 21%, breathlessness 20%, hematemesis 12%. oliguria 36% and anuria 6%26.

Fever was present in 44 (62%) of patients. Of these, 20% had diarrheal disease with fever, 18% had malaria, 18% had leptospirosis, 11% had sepsis, 5% had pyelonephritis. 25% had pyrexia of unknown origin (PUO) because the investigations were negative. PUO might most probably be malaria or leptospirosis because the QBC for malarial parasites is only 40% sensitive and MSAT (Macroscopic Slide Agglutination Test) becomes positive only the end of the first week of fever.

Vomiting occurred in 33 (46%) patients. Vomiting further complicated the malarial and leptospiral ARF by causing pre renal element. Giddiness and breathlessness occurred in 34% and 31% of patients respectively. Giddiness represented the hemodynamic compromise. Oliguria occurred in only 28% of the patients. The others did not complain of decreased urine output. Altered sensorium was present in 20% of patients, which was due to cerebral malaria, uremic encephalopathy and sepsis.

The most common sign was anemia, which was present in 34% of the patients. Most of these patients had malaria and gastrointestinal bleeding. The other signs are lung crackles 20%, elevated JVP 17%, ascites 10%, jaundice 8%.

The crackles were due to pulmonary edema and consolidation. Jaundice occurred in the patients with malaria and leptospirosis.

ACUTE DIARRHEAL DISEASE

Acute diarrheal disease was present in 11(16%) patients. The mean age of these patients was 50 years. All patients had diarrhea. 8 (73%) of patients had associated vomiting. 9 patients (82%) had fever. 5(45%) patients were dehydrated, hypotensive and oliguric. The mean B.urea was 71 mg% with the mean S.creatinine of 2.4 mg%. One patient had uremic symptoms and died.

MALARIA

8 (11%) patients had malarial fever. The mean age was 44 years. All had fever. Out of these, 5 (63%) patients had oliguria, headache, vomiting and were anemic. 4 (50%) patients had cerebral malaria and one patient had jaundice. The mean B. urea was 92 mg% with a mean S.creatinine of 4.2 mg%. One (13%) patient had P. falciparum infection and 7 (87%) patients had P. vivax infection. All were treated with quinine. One (13%) patient was started on hemodialysis as he had severe renal failure (S.creatinine-17.1 mg%) with uremic symptoms. The incidence of ARF in malaria varies from 1-60%.

M. Jayakumar et al shows that malarial ARF constitued to 4% of ARF, which was mostly due to falciparum[14]. RP Mathur and A Bhargav showed that falciparum caused 8% of ARF in their study[26].

LEPTOSPIROSIS

8 (11%) patients had leptospirosis. The mean age was 48. All the patients had fever. 5 (63%) patients had vomiting. 4 (50%) patients had severe myalgia. 3 (38%) patients had headache, diarrhea and were anemic and jaundiced. The mean B. urea was 75 mg% with the mean S.creatinine of 2.6 mg%. MSAT was ++ in 3 patients and +++ in 5 patients. MAT was positive in two patients and the serogroups were semoranga and australis. Semoranga is a non pathogenic strain.

Muthusethupathi et al showed in his study in 1991 that renal failure complicates leptospiral fever in 71% of patients and it was severe renal failure in 21% of patients[9]. Recent study (2005) shows renal failure in leptospirosis has decreased to 10%. M. Jayakumar et al study showed leptospirosis constituted 8% of ARF. They also showed a decline in leptospiral ARF from 31% in 1991 to 8% in 2004[14]. All our patients were treated with intravenous Crystalline Penicillin and C.Doxycycline.

CARDIAC FAILURE

8 (11%) patients with ARF had congestive cardiac failure. 2 patients had Rheumatic heard disease with atrial fibrillation and cardiac failure. The other six patients had Coronary artery disease with congestive cardiac failure. The mean age was 62 years. All patients had breathlessness. 7 (88%) patients had volume overload in the form of either pedal edema, facial puffiness, ascites

and raised JVP. 4 (50%) patients presented with pulmonary edema. The mean B. urea was 75 mg% with the mean S.creatinine of 2.0 mg%.

SEPSIS

Sepsis with ARF was found in 6 (8%) patients. The mean age was 58 years. 5 patients had fever. 4 patients were oliguric. 2 patients had altered sensorium. 3 patients had pneumonia, 2 patients had cellulitis, 1 patient had gluteal abscess. The mean B. urea was 78 mg% with the mean S.creatinine of 3.3 mg%. Sepsis contributed 9% of ARF in the study of Jayakumar M et al[14].

DRUGS

Drugs caused ARF in 6 (8%) patients. The mean age was 52 years. 4 patients had NSAID intake, one patient had aminoglycoside injectons and the other patient took native medicine for low back ache. The mean B. urea was 74 mg% with the mean S.creatinine of 3.5 mg%. The patient with native medicine induced ARF had uremic symptoms with S. creatinine of 7 mg% and was started on hemodialysis. Drugs caused 13% of ARF in the study of Jayakumar M et al. The common drugs were unknown analgesics, rifampin and NSAIDS[14,28].

Gastrointestinal bleeding was present in 3 patients. 2 patients had hemetemesis and one patient had malena. All were hypotensive.

3 patients had acute glomrulonephritis. All of these patients had facial puffiness, pedal edema, oliguria and hypertension. Two patients had hematuria and proteinuria. Glomerular disease oincluding crescentric glomerulonephritis, postinfective proliferative glomerulonephritis, SLE and IgA nephropathy accounted for 9% of ARF in Jayakumar M et al study[14].

Diabetic ketoacidosis occurred in 2 patients. One patient had Type 1 DM and the other had Type 2 DM. Both of these patients had severe vomiting. Their creatinine returned to <1.5 mg% on the third day.

2 patients had obstructive uropathy. One patient had Ca Bladder and was operated. The other patient had bilateral ureteric stones and was surgically managed.

One patient had alcohol induced rhabdomyolysis with myoglobinuria with positive benzidine test. He was started on forced alkaline diuresis. Rhabdomyolysis accounted for 0.6% of ARF in Jayakumar M et al study[14].

BLOOD PRESSURE

4% of patients had prehypertension. Stage I hypertension was present in 10% of patients. 15 (21%) patients had stage 2 hypertension. Of these 8 had systemic hypertension and 2 patients had AGN. The mean systolic B.P was 125 mm Hg. 9 (13%) patients had B.P <90/60 mm Hg. Of these, 5 patients had diarrhea and 3 had vomiting. One patient had CCF and one patient had hemetemesis.

ANEMIA

Anemia with hemoglobin <10 g% was found in 55% of patients. Of these, 15% had Hb <8 g%. These patients had malaria, leptospirosis and gastrointestinal bleeding. The mean Hb was 8.5 g%.

INVESTIGATIONS IN ARF

The mean B. urea was 79 mg%. 29 (41%) patients had B. urea in the range of 40-59. 16 (23%) patients had B. urea> 100 mg%. 10 (63%) of these patients had infections like malaria, leptospirosis, sepsis, diarrheal disease and PUO.

The mean S.Creatinine was 2.7 mg%. 53 (75%) patients had mild renal failure with creatinine <3mg%. 6 (8%) patients had severe renal failure with creatinine > 5 mg%. In the patients with severe renal failure, infective etiology was present in 4 patients, out of which 2 had PUO, one had malaria and one had sepsis. 2 patients had drug induced ARF and severe renal failure. Two patients were started on hemodialysis and one patient was put on peritoneal dialysis because of hemodynamic unstability. The patient who was put on peritoneal dialysis died.

The mean S. sodium was 137 mEq/L. 34% of patients had hyponatremia (S.Sodium < 135 mEq/L). 10% of patients had hypernatremia (S.Sodium >145 mEq/L). Hyponatemia was found common in ARF.The mean S.potassium was

4.1 mEq/L. 7 (10%) of patients had hyperkalemia (S.Potassim > 5.0 mEq/L). Of these 3 patients had leptospirosis, one had malaria, one had diarrheal disease, one had AGN and one patient had pyelonephritis.

Significant proteinuria was present in 9 (11%) of patients. 17(24%) patients had trace albumin which included patients with Type 2 DM, urinary tract infection, leptospirosis and malaria. 9 (12%) patients including type 2 DM and malaria had + urine albumin. 4 (5%) patients with PUO had ++ urine albumin. 4 (5%) patients with AGN and PUO had +++ urine albumin.

9 (12%) patients had ischemic changes in the ECG and they were known CAD patients. 11 (15%) patients had cardiomegaly in the Chest X-Ray. 6 (9%) had pleural effusion. 6 (9%) had pulmonary edema in the Chest X-Ray.

TREATMENT

The patients with ADD were treated with intravenous ciproflxacin, iv metrogyl and Intravenous fluids. Normal saline was used as a replacement fluid for vomiting. Ringer lactate solution was used for diarrhea.

Patients with malaria were given iv quinine. Patients with leptospirosis were treated iv crystalline penicillin and C. Doxycycline. Patients with sepsis were given high grade antibiotics and other supportive measures.

AGN was treated with salt restriction to < 2 g/d, fluid restriction to < 1L/d, Calories 35-50 kcal/kg/d mainly by carbohydrates, restriction of potassium intake, antihypertensives and diuretics.

Acute pyelonephritis was treated with sensitive antibiotics. DKA was treated with adequate fluids, antibiotics and Insulin. The offending nephrotoxic drugs were withdrawn. Forced alkaline diuresis was given for myoglobinuria.

RENAL REPLACEMENT THERAPY

Two patients were started on hemodialysis and one patient was started on peritoneal dialysis. All had severe hyperkalemia and S.creatinine >5mg% and uremia. Patients who were started on HD included malarial ARF and native medicine induced ARF. A patient with PUO was started on PD and he died.

MORTALITY

The mortality rate was 3%. It was low because most of the patients hadmild renal failure which was conservatively managed.

CHRONIC KIDNEY DISEASE

In our study, 82 (54%) patients had CKD. Of these 48 (59%) were males and 34 (41%) were females. The mean age was 55 years and range was 13-82 years. 80% of patients were above the age of 40 years.

ETIOLOGY

The most common cause of CKD was found to be Chronic glomerulonephritis 49%. 37 (45%) patients had Diabetic Nephropathy. 6 (7%) had obstructive uropathy. CGN also included the chronic interstitial nephritis since we dont have evidence to differentiate both of them.

Vikrant S and Kaushal SS reported in their study that the causes of CKD were - Diabetic nephropathy 64 (28.4%), Hypertension 40 (17.8%), CGN 20 (8.9%), CIN 95 (42.2%), ADPKD 5 (2.2%), and other 1 (0.4%)[29]. Dharan KS, John GT determined the etiological profile of chronic kidney disease (CKD) by analyzing the renal biopsies of patients with severe CKD. 70% of these patients had glomerulonephritis as the histological diagnosis. 12% had interstitial nephritis, 7% had hypertensive arteriosclerosis and 6% had metabolic nephropathies. This shows that the predominant cause of CKD is glomerular disease[30].

Acute on chronic CKD was present in five patients. Of these three had CGN and two patients had DN. Two patients had diarrheal disease, two patients had CCF with pulmonary edema.

The age and sex distribution showed that the mean age for CGN was 48 years and DN was 56 years. In CGN, 73% were males and 27% were females. In DN, majority were females (62%) and males were 38%. There were no patients with DN below the age of 34.

CLINICAL FEATURES OF CKD

Pedal edema was the commonest symptom which was present in 70% of the patients. Oliguria occurred in 54% of patients. The other symptoms were breathlessness 49%, facial puffiness 41%, giddiness 35%, vomiting, chest pain, dysuria, hiccups and hematuria.

Anemia was the most commonest sign which was seen in 57% of the patients..Lung crepititions was present in 30%. Ascites occurred in 21%.

43% of patients with CGN were known cases of SHT. 28% of patients were known CKD patients. The rest 72% were diagnosed at this present admission.

76% of the DN patients had history of DM and 35% had history of DM in the range of 1-5 years. 24% of the patients were diagnosed with DN with CKD at this present admission. Out of 82 patients with CKD, 37 (45%) had history of SHT.

Hypertension is an important risk factor for adverse cardiovascular outcome in patients with CKD[31]. In our study, 72% of CKD patients were hypertensive. 51% of the patients had Stage 2 hypertension (>160/100 mm Hg) which shows that control of hypertension is poor. 13% of patients had accelerated hypertension >210/130 mm Hg. Most of these patients had acute left ventricular failure. The mean BP was 152/92 mm Hg. Kumar V and Kaushal SS reported that 60% of patients with CKD were hypertensive[32].

INVESTIGATIONS

61% of the patients with CKD were anemic with Hemoglobin level< 10 g%. The mean Hb was 8.1 g%. The Hb level varied from 4-10 g%.

The mean B. Urea in the patients with CKD was 87 mg%. 35% of the patients had urea >100 mg%. The mean S.creatinine was 3.7 mg%. 23% of the patients had severe renal failure with S. creatinine >5 mg%.

The mean S. sodium was 139 mEq/L. 24% of the patients were hyponatremic and 18% were hypernatremic. The mean S. potassium was 3.9 mEq/L. 12% of the patients were hyperkalemic. Most of them with hyperkalemia had severe renal failure with S.creatinine of >5 mg% and uremic symptoms. Significant proteinuria occurred in 26% of patients with CGN and 43% of patients with DN. Kumar V and Kaushal SS reported that 80% of patients with CKD had proteinuria of varying degrees32.

Cardiovascular disease is the leading cause of morbidity and mortality in patients with CKD. 38% of the patients had ECG evidence of left ventricular hypertrophy. 16% of the patients had evidence of coronary artery disesase and ischemia. 6% of the patients had tall T waves and hyperkalemia. 53% of the patients had cardiomegaly in the X-Ray. 15% had pleural effusion and 5% had pulmonary edema. Kumar V and Kaushal SS showed 57% of patients with CKD had evidence of left ventricular hypertrophy and coronary artery disease in ECG32.

71% of the patients had kidney size < 9 cm. 40% of patients had kidney size in the range of 8-7 cm. 7% of the patients had hydroureteronephrosis. 22% of the patients had normal sized kidneys.

TREATMENT

5 out of the 82 CKD patients were taken up for the transplantation program because they had suitable donors and further evaluation was started.

Protein restriction of 0.6-0.8 g/kg/d of high biologic value protein was advised for the patients. No added salt diet and fluid restriction to output plus 500ml was suggested. Hypertension was aggressively treated with loop diuretics and calcium channel blockers. Potassium restriction was done. Anemia was treated with iron tablets because most of the patients had inadequate iron stores. Hemodialysis was started for 6 patients. 5 patients were put on peritoneal dialysis because they had cardiac complications like dilated cardiomyopathy.

The mortality was 1%.

SUMMARY

- 153 patients with S.Creatinine >1.5 mg% were analysed in our study. 71(46%) had ARF and 82 (54%) had CKD.
- The most common etiology of ARF was acute diarrheal diseases(16%), followed by malaria, leptospirosis, CCF, sepsis, drugs and AGN. Infective diseases contributed to > 50% of the ARF.
- The most common symptom of ARF was fever (62%), followed by vomiting, giddiness and breathlessness. Oliguria occurred only in 28% of the patients. The most common sign was anemia (34%). Jaundice occurred in 8% of the patients.
- Of all the common causes of ARF, malaria caused severe renal failure with mean S.creatinine of 4.2 mg%, followed by drugs and sepsis. Most of the patients with CCF were dyspneic. Anemia occurred commonly in malaria. Oliguria occurred commonly in ADD and malaria.
- 13% of patients with ARF had hypotension. 55% of the patients were anemic. The mean B. urea was 79 mg% and the mean S.creatinine was 2.7 mg%. 34% of patients had hyponatremia and 10% had hyperkalemia and 11% had significant proteinuria.
- 1 patient had peritoneal dialysis and 2 patiens had hemodialysis. All others were managed conservatively. Two patients who had severe renal failure and uremic coma died.

- 82 (54%) patients had CKD. The most common etiology of CKD was CGN(49%), followed by DN(37%) and obstructive uropathy. There was male preponderance in CGN and female preponderance in DN.
- Pedal edema was commonest symptom followed by oliguria(54%). Anemia was present in 57% of the patients. Volume overload was present in >20% of patients.
- 45% of patients had history of SHT and 20% were known cases of CKD. On examination 72% of the patients were hypertensive. 13% had malignant hypertension.
- 61% of CKD patients were anemic. The mean serum creatinine 3.7 mg% and 12% of the patients had hyperkalemia. 76% of patients with CKD had proteinuria in varying degrees.
- 38% of patients had evidence left ventricular hypertrophy in ECG. 53% of patients with CKD had cardiomegaly in Chest X-Ray. 71% had contracted kidneys.
- Hemodialysis was started for 6 patients. Out of these 5 were enrolled for renal transplantation program.. 5 patients were put on peritoneal dialysis. Mortality was 1%.

CONCLUSIONS

- ARF occurred in 46% of patients and CKD in 54% of patients.

- The commonest cause of ARF is Acute diarrheal diseases. Infections (malaria, Leptospirosis, sepsis) contributed to >50% of ARF. Most of the patients with ARF had mild renal failure, which contributed to the lesser requirement of dialysis and low mortality rate.

- The commonest cause of CKD was CGN (49%) followed by DN (45%). 72% of patients had SHT. 57% had anemia. Since the prevalence of DM is increasing day by day, early diagnosis of DN by routine screening and retarding the progression by using ACEI or ARB will decrease the incidence of ESRD patients.

- Early diagnosis of renal failure utilizing S.Creatinine $\geq$ 1.5 mg/dl was essential to diagnose and prevent complications of acute and chronic renal failure.

PROFORMA

Name: **Age:** **Sex:**

IP No.:

Diagnosis:

Clinical Features:

- Fever
- Myalgia
- Diarrhea
- Vomiting
- Giddiness
- Altered Sensoriun
- Headache
- Oliguria
- Dysuria
- Hematuria
- Pedal edema
- Facial Puffiness
- Ascites
- Anasarca
- Dyspnea
- Chest pain
- Hiccups
- Jaundice
- Elevated JVP
- Anemia
- Crackles
- Wheeze
- Hemiplegia
- Dehydration
- Pulse rate
- Blood Pressure

Laboratory Profile

- Complete Hemogram
- Blood Sugar
- Blood Urea
- Serum creatinine
- Serum Electrolytes
- Urinalysis
- Liver function tests
- ECG
- Chest X-Ray
- USG Abdomen
- QBC
- MSAT

Treatment

- IV fluids
- Diuretics
- Antihypertensives
- Antibiotics
- Peritoneal dialysis
- Hemodialysis

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MASTER CHART

S.No	Diagnosis	Age	Sex	Fev	My	Dia	Vom	Gid	ASen	HA	Olig	Dys	PE	FP	Asc	Ana	Brea	CP	DM	DN
1	ARF/AGE	70	M	1		1	1				1		1				1			
2	ARF/MALENA	50	M	1			1			1			1				1			
3	ARF/AGE	65	M	1		1	1	1			1									
4	ARF/AGE	19	M	1	1	1	1	1												
5	ARF/AGE	37	F	1		1	1	1												
6	ARF/AGE	45	M			1	1				1		1		1					
7	ARF/AGE	55	F	1	1	1	1	1			1			1						
8	ARF/AGE/LEPTO	29	M	1		1	1			1										
9	ARF/AGE/MALARIA/LEPTO/UREMIA	55	M	1	1	1	1				1									
10	ARF/AGE/R LEG CLLULITIS/DM2/SHT	45	F	1		1	1				1									
11	ARF/AGE/SHT/DM2/CVA/UREMIA	70	M			1		1	1										2y	
12	ARF/AGN	51	M								1		1	1		1				
13	ARF/AGN	37	M	1	1		1				1		1	1	1	1	1			
14	ARF/AGN/PT	60	F	1						1	1	1	1	1			1	1		
15	ARF/ALC POLYNEUROPATHY	50	M																	
16	ARF/ALCOHOLISM/RHABDOMYOLYSIS	28	M				1				1									
17	ARF/ATN/C MALARIA/PHT	60	M	1	1		1	1	1				1	1	1		1			
18	ARF/BA/CAD/DCMP/PUL EDEMA	58	M	1				1									1			
19	ARF/CAD/SHT/PUL EDEMA	70	M	1				1										1		
20	ARF/CER MALARIA	14	M	1		1	1		1											
21	ARF/COPD/COR PUL	54	M				1				1		1	1	1	1	1			
22	ARF/COPD/COR PUL/SHT	60	M	1													1	1		
23	ARF/CQ RES MALARIA	60	F	1	1		1	1		1	1	1					1			
24	ARF/CVA/OA/NSAID	41	M																	
25	ARF/CVA/SHT/DM2	68	F																2y	
26	ARF/CVA/SHT/PT/COPD	60	M					1												
27	ARF/DM1/DKA	26	M	1	1	1		1									1	1		
28	ARF/DM2/AGE	58	F	1		1	1												5y	
29	ARF/DM2/CERE MALARIA	65	F	1			1	1	1										4y	
30	ARF/DM2/CH PANCREATITIS	32	M				1												3y	
31	ARF/DM2/DN/CCF	70	M	1				1					1				1		6m	
32	ARF/DM2/DN/SHT/DKA	60	M				1	1					1		1		1		20y	2y
33	ARF/DM2/EPILEPSY/NSAID	55	M																5y	
34	ARF/DM2/SHR/CCF/CID	70	F				1										1	1	5m	
35	ARF/DM2/SHT/DN/CCF/GLUTEAL ABSCESS	65	M	1							1	1	1						3y	
36	ARF/DM2/SHT/LEPTO	60	F	1	1		1	1	1	1									15y	
37	ARF/DM2/SHT/PUO	60	M	1					1	1			1						2y	
38	ARF/DM2/SHT/VBI	68	M				1	1		1								1	4y	
39	ARF/DRUG INDUCED	35	M								1	1								
40	ARF/FEVER	88	M	1	1		1		1		1									
41	ARF/FEVER/PT	40	M	1															4y	
42	ARF/HEMETEMESIS	74	M				1	1												
43	ARF/ICH	70	M																	
44	ARF/LEPTOSPIROSIS	38	M										1	1						
45	ARF/LEPTOSPIROSIS	70	M	1	1				1											
46	ARF/LEPTOSPIROSIS	14	F	1	1		1	1	1	1	1						1			
47	ARF/LRI/UTI	46	M	1				1				1						1		
48	ARF/LVF/PUL EDEMA	8	F														1			
49	ARF/LVH/SHT	67	M			1	1				1						1	1		
50	ARF/MALARIA	30	M	1	1					1	1									
51	ARF/MALARIA	46	M	1													1	1		
52	ARF/MALARIA/UREMIA	21	M	1			1		1											
53	ARF/PEO	40	M	1					1			1					1	1		
54	ARF/PL EFFU/TCC RADICAL CYSTECTOMY	68	M														1	1		
55	ARF/PUO	45	F	1	1													1		
56	ARF/PUO	30	M	1	1															
57	ARF/PUO	38	M	1	1		1													
58	ARF/PUO	52	M	1	1		1			1	1		1	1	1					
59	ARF/PUO/DM2	35	M	1				1		1									7y	
60	ARF/PUO/SEIZURES	70	M						1											
61	ARF/PUO/VBI/C SPONDY	85	M					1	1											
62	ARF/PYOPNEUMOTHORAX	62	M	1														1		
63	ARF/R LEG CELLULITIS/MET ENCEPHALOPATHY	70	M						1											
64	ARF/RHD/CCF	42	F				1					1								
65	ARF/RHD/CCF	45	M				1						1		1		1			
66	ARF/SHT/CVA/KEROSENE POISONING/DM2 /AGE	49	M			1												1		
67	ARF/SHT/DM2/AGE/ OLD CVA	68	M	1		1												1	5y	
68	ARF/SHT/LVF	70	M					1			1		1				1	1		
69	ARF/SYM POLYARTHRITIS/OA/NSAID	45	F	1	1														3y	
70	ARF/TB	50	F					1									1			
71	ARF/UTI/AC PYELONEPHRITIS	49	F	1			1	1				1								

MASTER CHART

S.No	Diagnosis	Age	Sex	Fev	My	Dia	Vom	Gid	ASen	HA	Olig	Dys	PE	FP	Asc	Ana	Brea	CP	DM	DN
72	CKD/CGN	40	M				1	1			1		1				1			
73	CKD/CGN	38	F	1			1				1		1	1	1		1			
74	CKD/ OLD PT/R PL EFFU/CGN	65	F										1			1	1			
75	CKD/AS/AR/CGN	38	M										1				1	1		
76	CKD/BA/ALCOHOLIC/CGN	70	M						1				1							
77	CKD/CAD/SHT/CGN	71	M			1					1		1	1						
78	CKD/CAD/SHT/CGN	56	M					1												
79	CKD/CAD/SHT/CCF/DM2/DN	51	M								1		1				1	1		
80	CKD/CAD/SHT/PUL EDEMA/CGN	45	M								1		1				1	1		
81	CKD/CCF/OBS URO	58	M								1		1	1			1			
82	CKD/CGN/ACUTE ON CHRONIC/DM2/DN	39	M								1		1	1		1	1			
83	CKD/CVA/CAD/SHT/CGN	77	M					1												
84	CKD/CVA/SHT/CGN	70	M																	
85	CKD/DM/DN	46	F			1	1				1		1	1		1			10y	
86	CKD/DM1/DN	34	M	1	1		1	1					1				1		10y	
87	CKD/DM2/DL/SHT	60	F								1		1		1	1	1		3y	1y
88	CKD/DM2/DN	55	F								1		1	1					5y	2y
89	CKD/DM2/DN	55	F				1	1					1	1	1				12y	10y
90	CKD/DM2/DN META ENCEPHALOPATHY	65	F																3y	1y
91	CKD/DM2/DN/D FOOT	73	F	1							1		1						1y	
92	CKD/DM2/DN/OA HIP	65	F	1	1							1							2y	1m
93	CKD/DM2/DN/SHT/CVA	56	F				1	1			1		1				1		20y	4m
94	CKD/DM2/DN/SHT/D FOOT	60	M			1	1	1				1	1					1	10y	2y
95	CKD/DM2/DN/SHT/HYPOTHYROIDISM	57	M					1			1		1	1	1	1	1		5y	
96	CKD/DM2/DN/SOL LIVER	48	M	1							1							1	8y	1y
97	CKD/DM2/SHT	65	F																1y	6m
98	CKD/DM2/SHT	55	M				1		1		1		1	1					5y	2y
99	CKD/DM2/SHT	70	F				1				1							1		
100	CKD/DM2/SHT/CAD/HYPOTHYROIDISM	75	F										1	1					10y	
101	CKD/DM2/SHT/CCF	51	F				1	1			1	1	1	1	1			1	20y	
102	CKD/DM2/SHT/CCF/DN	50	F				1				1		1	1	1	1	1		17y	2y
103	CKD/DM2/SHT/CVA	62	F	1				1						1					10y	
104	CKD/DM2/SHT/DN	78	M								1		1	1	1	1	1		5y	2y
105	CKD/DM2/SHT/DN/DR/CCF/PUL EDEMA	38	F				1	1			1		1	1		1	1	1	8y	2y
106	CKD/DM2/SHT/OB URO	70	F						1			1							1m	
107	CKD/ESRD/DM2/DN	50	M		1		1				1		1	1	1		1		8y	5y
108	CKD/ESRD/PUL EDEMA/CGN	37	F										1	1	1	1	1			
109	CKD/ESRD/PUL EDEMA/CGN	76	F				1				1		1	1		1		1		
110	CKD/ESRD/PUL EDEMA/CGN	30	F				1	1			1		1	1	1		1			
111	CKD/FEVER/CCF/OBS URO	60	M	1	1	1			1		1		1							
112	CKD/LIMB GIRDLE DYSTROPHY/CGN	13	F																	
113	CKD/MPGN	18	F								1		1		1	1	1	1		
114	CKD/MULTIINFARCT DEMENTIA/CGN	70	M				1											1		
115	CKD/PNEUMONIA/CGN	50	M	1		1														
116	CKD/RA/PARKINSON/CGN	74	M				1						1							
117	CKD/RHT/CCF/AF/DM2/SHT/OBS URO	50	M								1		1		1		1		1y	
118	CKD/SHT/CGN	70	F	1				1									1			
119	CKD/SHT/CGN	70	M					1			1		1	1			1			
120	CKD/SHT/DM2/DN	63	M										1	1			1	1		3m
121	CKD/SHT/CGN	36	M					1			1		1	1			1			
122	CKD/SHT/CGN	35	M					1												
123	CKD/SHT/CGN	43	F					1			1		1		1		1			
124	CKD/SHT/CGN	21	M								1	1	1	1						
125	CKD/SHT/DM2/DN	55	F	1				1			1		1	1			1	1		
126	CKD/SHT/CGN	66	M					1									1			
127	CKD/SHT/CGN	50	M					1			1		1	1	1		1			
128	CKD/SHT/CGN	60	F				1		1		1		1		1	1				
129	CKD/SHT/CGN	49	M							1		1								
130	CKD/SHT/CGN	65	M	1			1						1							
131	CKD/SHT/CGN	45	M								1		1	1	1		1			
132	CKD/SHT/CAD/DM2/DN	62	F	1				1			1		1	1						
133	CKD/SHT/CAD/BPH/HUN/OBS URO	73	M																	
134	CKD/SHT/CCF/CGN	68	F					1			1		1				1			
135	CKD/SHT/CCF/URTICARIA/CGN	60	M		1			1			1		1				1			
136	CKD/SHT/COPD/DM2	60	M					1					1		1		1			
137	CKD/SHT/COPD/CCF/CAD/MD2/DN	65	M	1	1		1						1				1	1	3y	
138	CKD/SHT/CVA/CAD/CGN	72	M	1							1		1	1			1			
139	CKD/SHT/CVA/CAD/CGN	50	M					1												
140	CKD/SHT/DM2	65	F						1										3y	
141	CKD/SHT/DM2/CAD/LVF	78	F														1		12y	
142	CKD/SHT/DM2/CCF	50	F								1		1				1		7y	
143	CKD/SHT/DM2/DN/ D Foot	40	F				1					1	1	1					3y	2y
144	CKD/SHT/LVF/CGN	65	M					1					1				1			
145	CKD/SHT/PNEUMONIA/CGN	45	M	1				1			1		1	1			1			
146	CKD/U GASTROPATHY/CGN	45	M				1			1										
147	CKD/UTI/OBS URO	47	M	1								1	1	1						
148	CKD/UTI/OBS URO/RENAL CALCULUS	52	M																	
149	ACUTE ON CHRONIC CKD/AGE/CGN	31	M	1		1	1				1									
150	ACUTE ON CHRONIC CKD/DM2/SHT/DN	39	F				1	1			1		1	1			1			
151	ACUTE ON CHRONIC CKD/DM2/SHT/DN/PUL EDEMA	37	M								1		1	1			1			
152	ACUTE ON CHRONIC CKD/SHT/CVA/CGN	50	M																	
153	ACUTE ON CHRONIC CKD/SHT/GTCS/CGN	82	M					1			1		1	1						

MASTER CHART

S.No	SHT	CKD	CAD	PR	Sys	Dias	JVP	Ane	Dehy	Crk	Stk	Others	TC	Hb	S1	U1	5	Na1	K1	U Alb	SB	TP	Alb	USG RK
1				100	80	50				1				8.2	59	53	2.1	140	3.5	nil				normal
2				90	100	70		1					5700	6	93	85	2	119	3.8	*				normal
3				90	160	80							7500	10.4	90	82	2.6	137	3.8	nil				
4				100	80	60			1			seizures	6200	10.4	96	78	2.2	147	3.8	trace	1.4	5.6	2.9	
5				90	110	70			1					8.3	66	92	3.7	140	2.8	*				normal
6				90	210	80			1					9.1	68	45	1.6	142	3.6	nil				
7				82	100	70		1	1				8800	9.2	60	60	2.2	130	3.9	*				
8				72	120	80						NSAID	7200	11	56	41	1.9	152	4.6	nil	0.6	6.1		normal
9				90	130	90		1				hiccups/jaundice	6300	5.8	156	128	3.6	139	5.2	*	1			
10	2 y			70	120	80		1					6800	9.8	94	80	2.6	137	3	trace				MRD
11	2y			80	110	70			1				5600	10.8	231	131	4.8	131	5.3	trace				
12				90	140	100		1				NSAID	7000	11	89	72	2.9	148	5.7	***				12.3 x 3.7
13				90	160	100	1	1		1		wheeze		11	110	123	4.6	128	4.7	nil	1			11.7
14				90	160	100		1		1		h uria	7100	10.8	64	84	3.2	142	4.2	**				10.5 x 4.7
15				90	120	80							8000	11.8	900	53	2.1	142	4	nil	1	6.5		
16				82	110	70					1	benzi +	6100	10.8	180	72	2.4	148	4.3	trace	0.9	7.2	3.5	MRD
17				80	110	70		1					6300	9	124	50	1.8	134	3.4	nil	1.8	5.2	3.2	14.1 x 5.4
18				90	60	40	1		1	1			9200	11.8	90	43	1.6	137	4	nil				
19	1m			90	130	80				1			6200	10.4	180	115	1.5	136	3.1	trace				9.8 x 4.2
20				88	100	70						seizures	5200	10.6	68	67	3.1	140	3.8	*	0.8	7.8	4.8	normal
21	6y			86	130	90				1		wheeze	7000	10.2	60	119	1.4	129	3.2	trace				
22	5y			76	110	80						h uria		8.8	135	62	1.5	138	4	nil				MRD
23				100	110	70							7600	8.6	255	59	1.6	134	3.2	trace	1	5.3	3.6	
24	2y			77	150	100					1		6000	10.8	52	42	2	146	3.4	nil				normal
25	2y			90	150	100		1	1		1			10	76	58	1.6	139	4.8	nil				normal
26				90	100	70		1			1	seizures	5200	10.8	128	84	3.8	144	4.5	nil				9 x 3.4
27				86	110	70			1				5000	11.2	223	34	1.8	129	3.9	*				
28				88	150	80							5400	8	437	40	1.5	146	4.6	nil	0.7			
29				92	140	90		1					4800	8.5	80	84	2.4	130	4.3	trace				
30				80	130	90							6000	10.8	200	23	1.5	141	4.3	*	1.2	7.2	3.3	HUN
31				100	100	80		1	1				8000	7.6	270	164	3	133	4.3	trace				
32				84	160	90	1			1				9.3	198	93	1.8	126	4.1	****				
33				88	100	70			1			seizures/jaundice		9	277	48	1.9	142	4.2	trace				MRD
34	5y		1m	82	150	100	1	1		1			6800	8.2	98	87	1.8	136	3.8	nil				
35				90	170	100				1			5300	10.4	56	74	1.9	140	3.9	*	0.8	5.2	3	normal
36	10y			100	160	80		1					6200	10.2	101	51	2	136	4.6	nil	1	6.7	4.6	
37				112	150	100							9200	10.6	89	49	2.3	133	4.5	*				MRD
38	4y			76	130	80								11	164	37	1.7	140	4	nil				
39				86	150	90							5000	7.8	120	68	7	135	3.2	trace				12 x 6 HUN
40	10y			88	110	70		1					6200	6.9	128	146	4.2	133	4.9	**	1.5	7.2	4.2	10.2 x 5.6
41				90	80	60		1				p uria/jaundice	8800	8	355	49	1.3	126	2.3	nil	0.8	6.9	3.7	
42				120	90	60		1				ulcer/dyspepsia	6100	10	146	106	2.1	128	3.1	nil	0.9	5.9	3.8	normal
43	10y			74	170	130					1			9.8	64	111	1.9	142	3.6	nil				
44				88	120	80						jaundice	6000	10	72	45	2	140	35	trace	1.6	7	3.2	normal
45			1y	100	110	60			1			jaundice	7200	7.8	68	128	4	128	5.8	nil	5.6	5.8	2.8	normal
46				80	80	60							2800	6.8	184	84	2.2	140	5.2	nil	1	5		10.1 x 4
47				94	110	80						NSAID		8.6	118	49	3.5	140	3.6	trace				
48				150	150	90	1			1				10.2	64	72	2.2	144	4.6	*				9.8
49				92	210	130				1			6500	10	118	57	1.5	124	2.7	trace				
50				100	110	70		1						11.4	60	52	1.9	140	4.2	**				MRD
51				90	120	80							6000	8.4	96	67	1.7	133	3.6	nil	1	7.3	3.5	
52				96	150	90						h uria	7350	8.8	125	288	17.1	151	4.6	**	1			normal
53				80	100	60		1	1			h uria	8900	6.5	143	273	9.4	138	3.7	nil	0.7			
54				100	90	70				1			7000	9	105	41	1.8	136	3.5	trace	1	6.6	4.2	9.2 x 3.6
55				86	90	60			1				5000	9	129	80	2.1	138	33	trace	0.6	5.9		
56				80	100	60							7200	10.2	146	101	1.5	140	4.2	trace	1.1			
57				90	100	70			1					10.2	60	51	3.1	138	4.1	***				
58				102	100	60		1				hiccups/jaundice		10.9	70	136	5.9	130	4.8	nil	4.9			
59				88	120	90				1		wheeze		7.8	226	36	1.6	142	3.6	**				
60	2y			90	140	90						seizures	4200	8.8	116	56	1.6	129	3.9	*				
61				90	150	90							7000	11	104	50	1.5	138	3.8	nil				
62				82	110	70		1	1			COPD	5250	9	97	47	2.1	129	3.8	**	1.2	6.2	3	normal
63	4m			92	200	120		1						11	154	126	5.4	118	5.5	**	0.8			MRD
64				80	110	70								10.8	120	54	1.8	139	4	nil				
65				110	130	80	1	1					9700	9.4	76	56	1.8	136	4.4	nil				
66	1y			90	140	90				1			9200	10.4	60	48	1.8	144	4.2	nil				
67	3y			80	200	120							6400	9	240	56	1.5	139	3.8	nil				
68	2y			100	210	100							5800	10	136	71	2	130	3.1	nil				
69				90	120	80							6900	10.2	120	142	5.5	132	5	**	1	7.2		
70				102	80	60		1					8200	9	198	54	1.8	135	4.7	trace	1	7.1	3.9	
71				90	110	80		1	1			UGI-N	6200	9	121	52	1.8	137	51	nil	0.9			10.8 x 4.6

MASTER CHART

S.No	SHT	CKD	CAD	PR	Sys	Dias	JVP	Ane	Dehy	Crk	Stk	Others	TC	Hb	S1	U1	5	Na1	K1	U Alb	SB	TP	Alb	USG RK
72				100	210	140		1					5800	10.9	68	128	6.2	136	3.3	*	1	7.6	3.3	8.4 x 3.7
73		2m		90	130	80		1					7280	8.5	62	65	2.7	131	4.8	nil				9.4 x 3.7
74				80	110	70		1						8.2	116	108	2.4	132	5.4	nil				8.2
75				70	200	100								9.2	62	98	4.6	149	4.8	*				8.4 x 3
76				74	90	60				1		wheeze	6100	9.5	76	60	2.4	144	4.2	nil				9.2
77	2y			74	170	90		1						8.5	107	78	3.5	138	4.2	*				9 x 4.3
78	2y		2y	80	140	90							8200	10.2	90	69	3.4	134	4	*				9 x 5
79	5y			140	220	110								8.8	180	76	2	140	3.8	trace				8.2 x 3.9
80	1y		1y	84	160	110	1							9.4	66	121	7.9	142	4.5	nil				7.6
81				88	140	80							7900	9.7	82	89	2.8	149	4.4	nil				hydronephros
82				90	130	90		1		1				10.8	198	231	11.4	140	3.2	*				8.2
83	5y			100	110	70				1	1		6400	11	114	71	2.2	149	3.6	***				10 x 4.6
84	6m			92	210	110					1			8.4	84	57	1.7	138	3.2	trace				7 x 4.5
85				89	170	100		1					9200	8	181	62	4.3	138	4.4	trace	1			6.8
86				96	180	90		1		1				8	107	78	3.2	145	5	*				8.8
87				90	210	80		1		1		jaundice	5900	9	64	98	5.8	142	2.9	*				10 x 4.6
88				90	130	80		1						8.6	273	78	1.6	146	3.8	**				10.1 x 4.6
89		5y		90	150	70		1					9200	11.8	82	165	11.7	151	5.9	**				9.2
90				82	110	70		1	1				6600	10	275	107	3.7	138	3.9	*				7.6 x 3.1
91				100	180	90							8200	11	108	56	1.6	142	3	**				8.8
92	2y			80	150	90			1	1			7600	8.4	128	120	2.5	134	3.5	**				MRD
93	12y	3m		80	160	100		1			1		7600	9.8	340	71	3.3	140	3.6	***			2.9	10.1 x 4.6
94	2m	2y		120	140	90	1	1	1			p uria/hiccups		7.8	93	77	2.1	142	4.9	*				MRD
95	6y	4y		90	130	80	1	1		1			8200	7.4	183	95	7.5	130	4.6	*	0.5	8.4	2.8	9.1
96				72	90	60		1					7100	9.6	171	69	2.4	139	4.6	**				MRD
97	1y			90	190	100			1			seizures		10.2	224	60	1.8	139	3.1	*				MRD
98	5y			80	170	80							7100	10.6	324	60	1.6	150	2.2	trace				10 x 4.6
99				80	150	80		1						6.2	50	77	2.2	143	4.5	*				10.1 x 4.6
100	10y			90	170	90		1					6000	8.4	217	59	4.1	137	3.7	*				6.6
101	2y		1y	92	140	90	1			1			6300	7.2	200	104	2.2	139	3.2	***				9.8
102	1y			84	150	100		1					5000	10.4	176	79	1.7	126	3.8	**		7	3.8	9.6
103	2y			70	210	120							4500	9	140	56	1.6	142	4.2	trace				8.3 x 4
104	2y			100	160	100	1			1		hiccups	4800	12	109	121	4.2	132	3.5	****	1			10 x 5
105	8y			86	100	70							8600	8.7	180	135	4.6	137	4	*	0.6	6.4	3.8	normal
106				84	140	90		1	1			h uria		8	162	157	6.8	123	3.2	**	1	7.1	4.2	10.1 x 5.2
107	5y			86	130	90		1		1			5000	6	104	77	7.2	151	5.5	trace		6.4	3.6	8.8
108		2y		102	170	100		1		1			7400	10.4	76	191	6.7	131	4.5	nil	0.8			6.5
109	5y	1y		80	180	120				1			9200	9.8	120	94	3	142	4	nil				7.8
110		8m		110	140	90	1	1		1			3600	4	81	221	15	122	6.1	nil				6.7 x 4.7
111			1m	106	100	70	1			1			6900	8	107	90	2.5	138	3.3	*	1	5.9	3.9	MRD calculus
112				78	110	80							8300	7	69	89	2.2	150	3.9	nil	0.9			7.4 x 3.2
113	6y	6y	2y	100	100	80	1			1			9200	8	112	52	1.7	136	4.3	nil		5.7	3.6	normal
114				82	110	70		1					7200	9	128	103	2.5	114	2	*				8.2
115			3y	80	100	60		1	1			gara	8100	10.2	62	201	4.7	138	3.8	nil				10.2
116				82	130	90		1				hiccups		9.8	72	146	2	138	3.8	**				7.4 x 3.2
117				120	220	110	1						6700	9.2	196	53	2.1	147	2.9	nil				HUN
118	2 m			90	210	120							6250	7	122	100	4.2	130	4.5	trace				9 x 3.1
119	2m	2m		80	140	90		1				hiccups		8.6	120	68	3	142	3.8	*				8.8
120	6m			90	180	100		1						8.1	94	140	46	122	4.2	*				7.3 x 3.8
121				86	150	80		1		1				10.4	98	142	5.9	139	4.9	trace	1			10
122				90	200	140								9.8	90	117	7.9	137	3.5	**				10.5 x 3.9
123				86	200	100	1			1			5600	9.2	113	102	2.3	142	4.7	**				9.3 x 4.2
124				96	140	100		1					6200	8	99	104	6	142	4.6	***	0.9	5.7	3.7	8.2 x 3.8
125	6m	6m		100	200	120		1		1			7200	10.2	172	84	2.4	130	4.5	nil	1	6.4	4	4.4 x 2.7
126				96	180	130	1	1		1			7100	8.2	140	84	2.2	144	4.6	nil				7.7 x 3.3
127				92	170	100		1					6450	7.8	82	175	9	140	3.5	nil				12 x 6
128	6y			78	150	90		1		1			6900	8	64	60	2	142	4	nil				7.5 x 3.4
129				80	130	80							7100	10	80	84	1.6	140	4	trace	1			5 x 2.4
130		6y		72	170	100		1		1			8400	8	136	123	3.9	146	3.5	trace	1			9.3 x 4.2
131		2y		100	180	100		1					6100	8	190	210	13.2	148	6.7	**				7.5 x 3.4
132				74	160	90	1	1						7.2	97	142	7	150	5.2	*				9.3 x 4.2
133				74	160	70							6000	7	131	75	1.6	152	4.2	*	0.8	6.7	4.6	9.2 x 5.2
134	10y			90	160	100			1				6500	9.2	138	49	2.3	136	3.5	trace		7.6	4.8	7.4 x 3.6
135				76	170	80		1					6100	9	109	58	2.6	149	6.4	**				7.4 x 3
136				120	220	170		1				wheeze	4800	10.2	48	44	4.1	139	4.1	*				10.5 x 4.4
137			6m	108	150	100							5800	9.6	276	70	2	133	3.2	**				MRD
138	15y	6m		90	160	100		1		1			8900	10.8	100	62	2.6	135	4.8	trace				MRD
139	6y	4y		90	130	80		1			1		6900	7.8	108	92	2.2	140	3.8	nil				8.2 x 3
140	5 y			80	230	100								6.8	97	92	2.6	134	4.2	*				9.3 x 4.2
141	12y		2y	80	160	100		1	1	1			6300	8.4	69	31	1.6	140	3.2	**				normal
142	4m			80	140	90					1			9	160	76	2.3	138	3.6	*				10.2
143	7y			80	170	90		1	1					10.2	104	40	1.2	136	3.8	*				6.8
144	6m	6m		100	190	60	1	1		1		p/h uria		8.8	90	132	8.6	138	6.2	**	0.9			7.5 x 3.4
145	2y			106	180	100	1	1						9.2	92	60	2.4	144	4.2	trace				8.2
146				88	120	80						UGI-N	7400	10.2	101	54	2.2	136	4.1	**				6.8 x 3.3
147				86	130	90						siddha	6800	11.9	96	82	3.8	132	5.1	nil	0.8	7.5	4.4	7.3 x 4.4
148		1y		90	100	60		1					8800	8	104	116	9.8	139	4	nil				HUN
149				80	100	70	1					wheeze		10	88	145	7.5	142	3.5	****				11.2 x 5.3
150				90	100	70								8.8	80	52	1.5	128	3.4	**				8.8
151				84	170	130		1				hiccups		7.8	120	60	1.6	134	4	trace				7.8
152	2y			80	170	90		1	1				7600	10.4	110	82	2.2	128	3.8	*				7.5 x 3.4
153	5y			80	180	100				1		seizures	8000	10.6	69	62	2.5	150	5.4	nil				8.7 x 3.1

MASTER CHART

S.No	USG LK	CXR	ECG	QBC	MSAT	Treatment	PD	HD	D
1	normal	cardiomegaly	WNL			cipro/metro/dopamine			
2	normal	PT	WNL			lasix/CQ/doxy/para/rani			
3		NAD	old ASMI			cipro/metro/rani			
4		NAD	WNL	neg		cipro/metro/rani/CQ/doxy/dom			
5	normal	NAD	WNL			cipro/rani/metro/doms			
6		NAD	WNL			cipro/rani/metro/doms			
7		NAD	old IWMI			cipro/metro/para/rani			
8	normal	COPD	CAD	neg	2+	cipro/metro/rani/para/doms			
9		NAD	WNL	PF	3+	CQ/para/rani/CP/dom/cefo/falci			
10	MRD	NAD	WNL			Cipro/Metro/IVF			
11		pl effu	poor prog R wave			cipro/metro/rani/cal			1
12	11.5 x 5.4	NAD	WNL			lasix/cefo/rani			
13	11.7	cardiomegaly	WNL			para/rani/amit/mixtard			
14	10.8 x 4.7	R LL pneumonia	inf wall ischemia			cefo/rani/doxy/nify			
15		cardiomegaly	CAD			bct/pari/rani/amtri/mixtard			
16	MRD	pul edema	LVH			ampi/deri/lasix/ceftri/metro			
17	10.4 x 5.5	NAD	WNL	posi	neg	Q/para/ald/lasix/ceftri/metro/rani/blood			
18		NAD	LAFB RBBB LID			ampi/deri/lasix/isdn/aldac/envas/asp			
19	10.8 x 3.8	cardiomegaly	lat wall ischemia			amlo/lasix/isdn/cefo/para/rani			
20	normal	NAD	WNL	neg	neg	cefo/quinine/para/rani			
21		COPD	sinus tachy/RAD			lasix/amox/rani/deri			
22	MRD	cardiomegaly	p pul			cipro/rani/deri/lasix/bct			
23		NAD	WNL	PV		Q/para/doxy/cipro/rani/doms			
24	normal	NAD	LVH with strain			lasix/rani			
25	normal	NAD	WNL			cefo/rani/deri/asp/bct			
26	9.5 x 4.2	R UL pneumonia	WNL	PV	3+	CP/metro/ATT/amlo/asp			
27		cardiomegaly	sinus tachy			cipro/metro/rani/omez/HA			
28		L pl eff	sinus tachy		3+	cfo/glipi/para/cq			
29		bronchiectasis	sinus tachy			quinine/cefo/para/rani			
30	HUN	R LL cystic changes	WNL			cipro/rani/metro/doms			
31		NAD	old IWMI			cipro/cefo/metro/rani/H A/lasix/envas/asp/isdn			
32		cardiomegaly	WNL			ceftri/metro/doxy/lasix/rani/amlo/envas/insulin			
33	MRD	NAD	WNL			pheny/mnni/lasix/cefo/rani/para/HA			
34		cardiomegaly	LVH with strain			ampi/aten/dom/lasix/envas/isdn			
35	normal	B pl effu	WNL			ceftri/para/rani/amlo/dom/met/gli			
36		cardiomegaly	WNL		2+	CQ/doxy/qui/amlo/rani/cefo/HA/dom			
37	MRD	bronchiectasis	WNL	neg	2+	ceftri/para/rani/amlo/dom/met/gli			
38		WNL	CAD			ampi/amlo/lasix/isdn/insulin			
39	9.4 x 5.3 HUN	pneumonia	WNL			ceftaz/lasix/cipro/rani		2	
40	9.8 x 4.5	NAD	WNL			doxy/para/CQ/dos/amlo			
41		NAD	WNL	neg	neg	doxy/cipro/HA/CQ			
42	normal	NAD	IRBBB RAD			panto/blood/amox/metro/cefo/rani/meto			
43		patchy infil	old ASMI			cipro/para/rani/Q/CP/asp			
44	normal	NAD	LVH		3+	cp/rani/para/bct			
45	normal	cardiomegaly	old ASMI	neg	neg	CP/cefo/metro/panto/hepa/vitk/Q/rani/asp/isdn			
46	9.2 x 4	pul edema	WNL	neg	3+	cipro/para/CQ/rani/dom/qui/ceftri/acyclovir			
47		patchy infil	P pulm LVH			cipro/metro/amlo/rani			
48	10.6	pul edema	sinus tachy			CQ/cipro/doxy/rani/para/dom			
49		NAD	WNL			CQ/cipro/doxy/rani/para/dom			
50	MRD	NAD	LVH			Q/omez/para/doxy/pheny			
51		NAD	WNL	neg	neg	cipro/doxy/CQ/rani/para/dom			
52	normal	NAD	WNL		neg	qui/omez/para/doxy/pheny		1	
53		NAD	LVH with strain			CQ/cipro/doxy/rani/para/dom			
54	9 x 4	L pl effu	WNL			dopamine/aten/asp/rani/cefo			
55		pl effu	LVH	neg	neg	CQ/para/rani			
56		cardiomegaly	WNL	neg		CP/cipro/rani/para/deri/cefo/			
57		cardiomegaly	WNL			CQ/para/rani			
58		NAD	WNL	neg		qui/doxy/CP/rani	1		1
59		NAD	WNL			HA/cefo/para/rani/deri			
60		bronchiectasis	RAD RBBB			pheny/rani/deri/amlo/lasix			
61		NAD	WNL	neg		seftri/CQ/para/asp/rani/amlo			
62	normal	hydropneumothorax	LVH			lasix/asp/isdn/			
63	MRD	NAD	tall T waves			dig/lasix/rani/deri/cefo/acitrom			
64		cardiomegaly	sinus tachy			lasix/asp/envas/aldac/KCl			
65		cardiomegaly	P mitrale/AF			dig/lasix/rani/deri/cefo/kcl/acitron			
66		pl effu	LVH with strain			cefo/rani/metro/asp/lasix/amlo/NTG			
67		NAD	LVH with strain	PV		doxy/Q/CQ/rani/dom/amlo/prima/asp/metf/glipi			
68		NAD	CLBBB LVH with strain			isdn/envas/rani/cefo/lasix			
69		NAD	WNL			rani/tramadol/predni/para			
70		PT/ILD	p pul/ poor prog R wave			cefo/rani/deri/cipro/E/liv52			
71	10.9 x 5.8	NAD	WNL			rani/cipro/oflox/doms			

MASTER CHART

S.No	USG LK	CXR	ECG	QBC	MSAT	Treatment	PD	HD	D
72	7.3 x 3.8	cardiomegaly	LVH tall T waves			lasix/rani/nife/cal/			
73	6.8 x 3.2	NAD	WNL			lasix/amlo/cal			
74	7.8	cardiomegaly	LAD LAFB old AWMI			cipro/rani/cefo/amlo/cal/lasix			
75	7.8 x 3	cardiomegaly	LVH with strain			amlo/rani/lasix/cal/isdn/asp/ald			
76	8.8	pl effu	WNL			cefo/rani/cal			
77	8.7 x 4.7	NAD	IWMI			lasix/cefo/amlo/rani/isdn/cal			
78	9.9 x 4.4	cardiomegaly	LVH			asp/amlo/aldom/isdn/aten/cal			
79	9.4 x 4.2	cardiomegaly	sinus tachy/LVH with strain			lasix/amlo/rani/amox/aldom			
80	6.7	cardiomegaly	LVH			lasix/ampi/rani/amlo/cal/asp		1	
81		pul edema	WNL			ampi/deri/rani/asp/lasix			
82	10	cardiomegaly B pl effu	LVH			lasix/deri/rani/nife cal		1	
83	10.4 x 5.1	cardiomegaly	IWMI			lasix/nife/NTG/deri/rani/cefo			
84	5.4 x 2.5	cardiomegaly	LVH			lasix/mannitol/asp			
85	7.2	NAD	LVH			lasix/rani/cal/doms/amlo			
86	8.4	cardiomegaly	sinus tachy	neg		cefo/rani/amlo/cal/heparin/lasix			
87	10.4 x 5.1	pul edema	tall T waves			lasix/cal/amlo/ampi/rani			
88	10 x 4.9	NAD	WNL			mix/glimi/lasix/cipro			
89	9	NAD	WNL			lasix/amlo/rani/doms			
90	8.2 x 2.9	COPD	sinus tachy			ampi/rani/glipi/cefo/HA			
91	8.6	cardiomegaly	poor prog of R wave			cefo/metro/asp/isdn/envas/lasix/amlo/HA			
92	MRD	cardiomegaly	poor R wave prog			lasix/cefo/rani/amlo/HA			
93	10 x 4.9	NAD	LVH			cefo/ha/amlo/lasix/cal			
94	MRD	NAD	LVH			cefo/metro/rani/doms/amlo			
95	9.3	cardiomegaly	low voltage complexes			lasix/deri/cefo/cal/rani/amlo/HA			
96	MRD	pl effu	LVH			cefo/rani/deri/cal/lasix/HA/I&D			
97	MRD	pneumonia	sinus tachy			lasix/rani/amlo/cal/OHA			
98	10.4 x 5.1	cardiomegaly	sinus brady			cefo/rani/amlo/OHA/para/asp			
99	10 x 4.9	cardiomegaly	LVH			rani/cepha/amlo			
100	6.8	cardiomegaly	LVH			amox/nife/pheny/eltrox/rani/asp/cal			
101	10.2	pul edema	sinus tachy			dig/lasix/norf/rani/HA/amlo/asp/hep			
102	9.2	cardiomegaly	WNL			insulin/amlo/aldo/lasix			
103	8.4 x 3.6	NAD	LAFB LVH with strain			amlo/amox/rani/lasix/cal/HA			
104	10.5 x 5.3	cardiomegaly/pul edema	poor prog R wave			cipro/cefo/metro/lasix/insulin/ amlo/isdn/asp	1		
105	normal	pneumonia	WNL			lasix/aten/amlo/dig/cal/HA			
106	9.4 x 5	cardiomegaly	WNL			cefo/metro/amlo/actrapid			
107	7.8	cardiomegaly	CAD			lasix/amlo/aldom/rani/glipi	1		
108	7.6	NAD	WNL			lasix/ampi/aten/amlo/aldo/rani			
109	7.6	cardiomegaly	lat wall ischemia			lasix/amlo/aten/cal/dom/rani			
110	7.4 x 3.1	pl effu	CAD			lasix/deri/ampi/cal/amlo	10	1	
111	MRD	cardiomegaly	low voltage complexes	neg		dopamine/CQ/cefo/cal/rani/deri/para/metro/qui/isdn			
112	7.2 x 3.2	NAD	WNL			lasix/cal/rani			
113	normal	cardiomegaly	LVH			rani/deri/cefo		28	
114	8	NAD	VPD			ampi/rani/bct			
115	10	R LL pneumonia	WLL			cefo/cipro/amlo/rani/lasix			
116	7.4 x 3.1	cardiomegaly	WNL			cefo/rani/syndopa			
117	HUN	R pl effu	AF			amox/rani/dig/kcl/aldac/lasix			
118	9.2 x 39.2 x 3	R LZ Infiltrations	WNL			ampi/lasix/amlo/cal/aten			
119	8.2	NAD	LVH with strain			lasix/amlo/asp/isdn/rani/domp/cal			
120	8 x 3.5	cardiomegaly	LVH with strain LAD			amlo/anox/rani/aldo/lasix			
121	9.4	cardiomegaly	LVH with strain			amlo/cal/rani/cal glu iv			
122	10.4 x 5.1	cardiomegaly	LVH with strain			lasix/amlo/aldom/rani/cal			
123	8.8 x 4.2	B pl effu	low voltage complexes			lasix/cal/amlo/aldom			
124	8.1 x 3.2	NAD	WNL			doxy/para/rani/amlo/ca			
125	8.1 x 4.8	cardiomegaly pul edema	LVH			cefo/lasix/nife/aldo/rani/aten/ca			
126	7.7 x 4.2	cardiomegaly	LVH			lasix/rani/cal/amlo			
127	12 x 6.2	NAD	LAE LVH with strain			cefo/lasix/amlo/aten/rani/cal			
128	7.4 x 3.7	NAD	non prog of R wave			amlo/aten/lasix			
129	7.8 x 4	cardiomegaly	LAE			lasix/nife/cefo/rani			
130	8.8 x 4.2	cardiomegaly	Old ASMI			ampi/lasix/deri/amlo/rani/cal			
131	7.4 x 3.7	cardiomegaly	LAE			lasix/HA/calglu/rani/amlo		1	
132	8.8 x 4.2	P Effu	old asmi			amlo/isdn/asp/lasix	1		
133	9.4 x 5.2	cardiomegaly	WNL			lasix/nife/isdn/asp/dig/amox			
134	7.8 x 3.5	cardiomegaly	lat wall ischemia			lasix/amlo/rani/isdn			
135	6.8 x 3.8	cardiomegaly	WNL			alben/amlo/lasix			
136	6 x 3.2	B pl effu	LAD LVH			cefo/rani/deri/cal/nife/aldo/aten			
137	MRD	cardiomegaly	inf wall ischemia	neg	neg	cipro/doxy/isdn/envas/dig/lasix/asp/rani/dom			
138	MRD	cardiomegaly	LVH with strain			lasix/doxy/nife/aldo/isdn/rani/CQ	1		
139	9.2 x 2	cardiomegaly	old IWMI			amlo/asp/isdn/rani/amox			
140	8.8 x 4.2	cardiomegaly	WNL			ampi/rani/amlo/aten/glipi			
141	normal	cardiomegaly	LVH old ASMI			lasix/isdn/asp/rani/envas/NA			
142	10.6	R pl effu	LVH with strain			lasix dig aldac cefo			
143	7.2	NAD	WNL			cefo/metro/envas/asp/nife/rani/trental/insulin			
144	7.4 x 3.7	cardiomegaly B ple effu	LVH tall T waves			lasix/rani/cal/amlo			
145	8.6	R LL pneumonia	sinus tachy			cefo/lasix/rani/deri/amlo			
146	7.7 x 3.3	emphysema	LVH			ampi/rani/cal/onez/doms			
147	8.1 x 3.6	NAD	tall T waves			cipro/rani/cal/amlo			
148	HUN	NAD	WNL			cipro/cal/rani		1	
149	11 x 5.6	cardiomegaly	LVH			lasix/cipro/metro			
150	9	pl effu	WNL			ampi/rani/cal/deri			1
151	7.2	NAD	lat wall ischemia			lasix/nife/HA/isdn/			
152	7.4 x 3.7	cardiomegaly	tall T waves			cefo/cipro/metro/rani/lasix/asp/amlo			
153	7.9 x 4	NAD	LVH with strain			pheny/lasix/rani/amlo/cal			

FIGURE 1

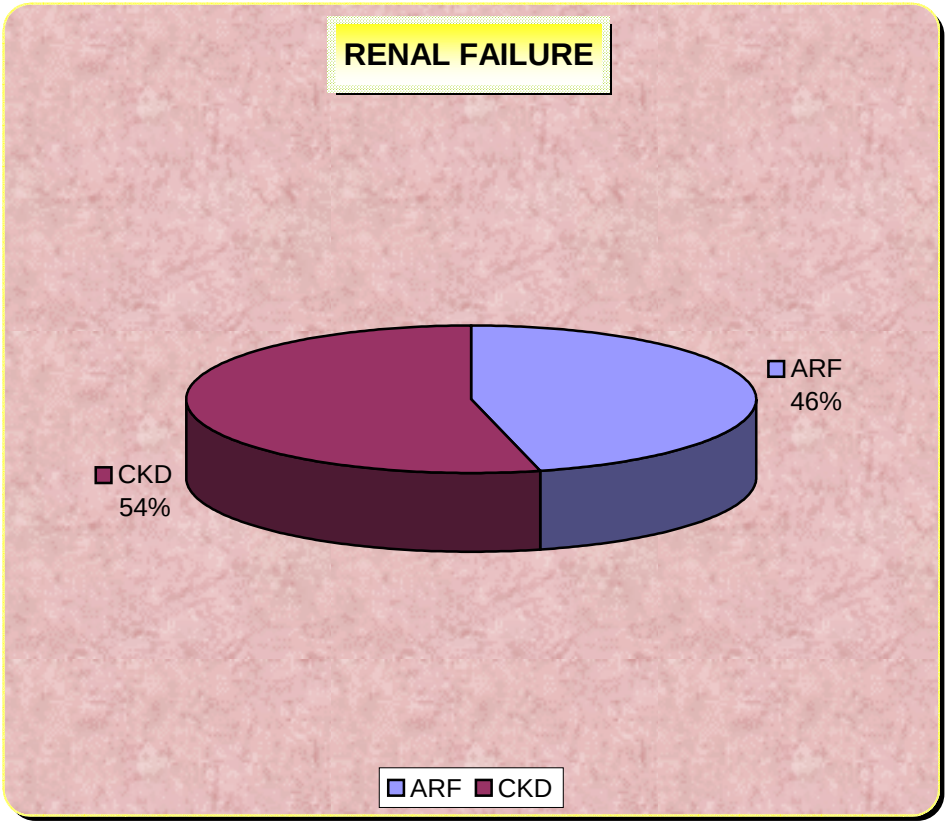


FIGURE 2

ETIOLOGY OF ARF WITH FEVER

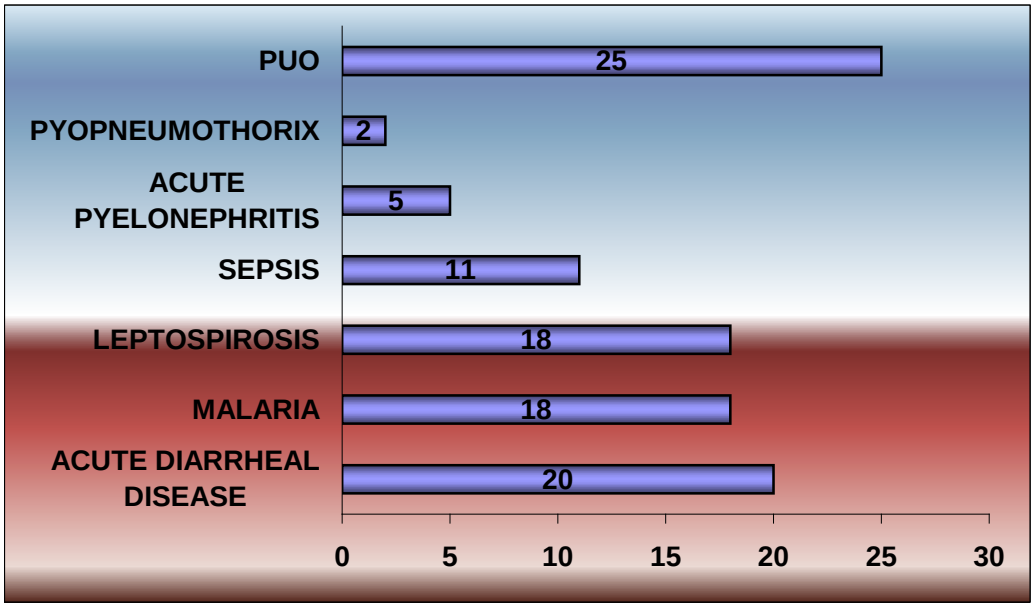


FIGURE 3

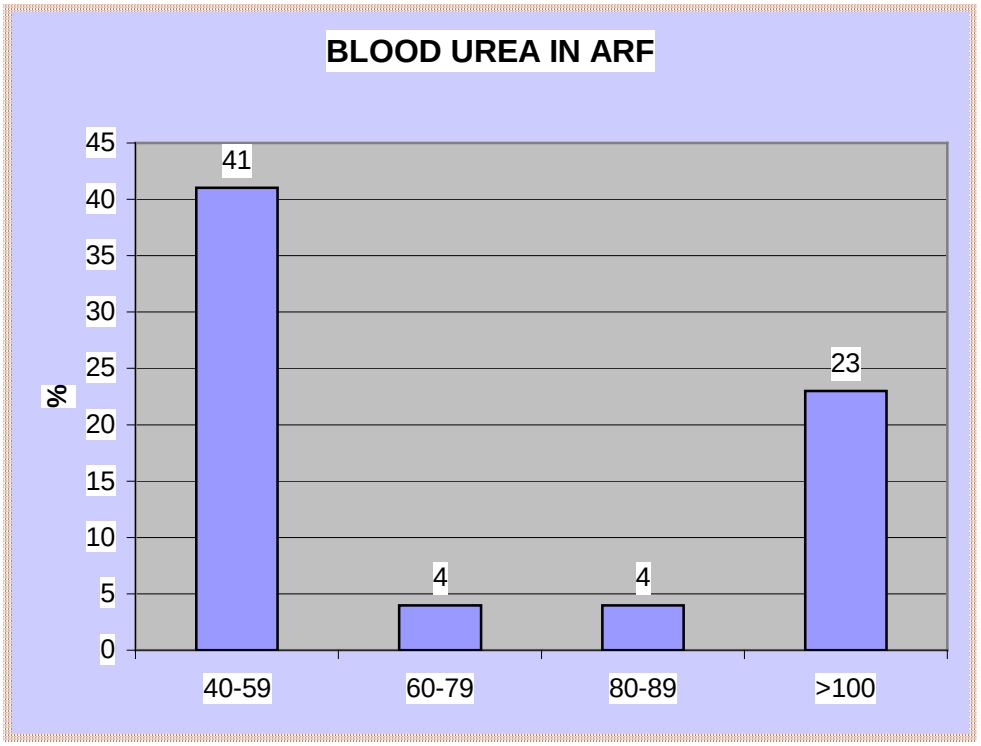


FIGURE 4

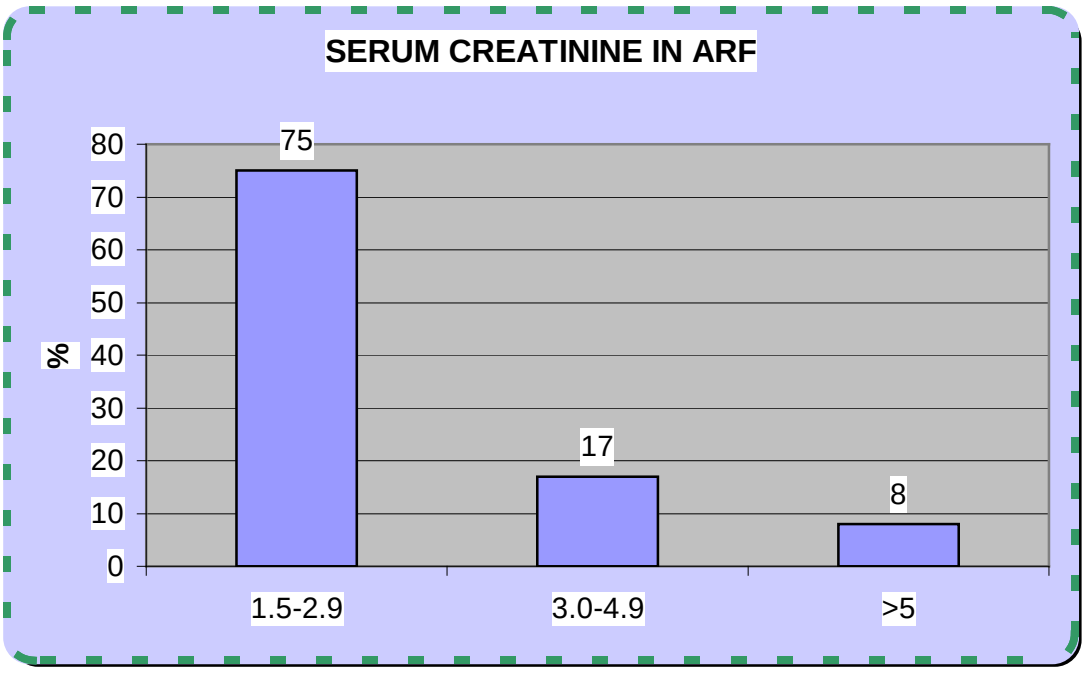


FIGURE 5

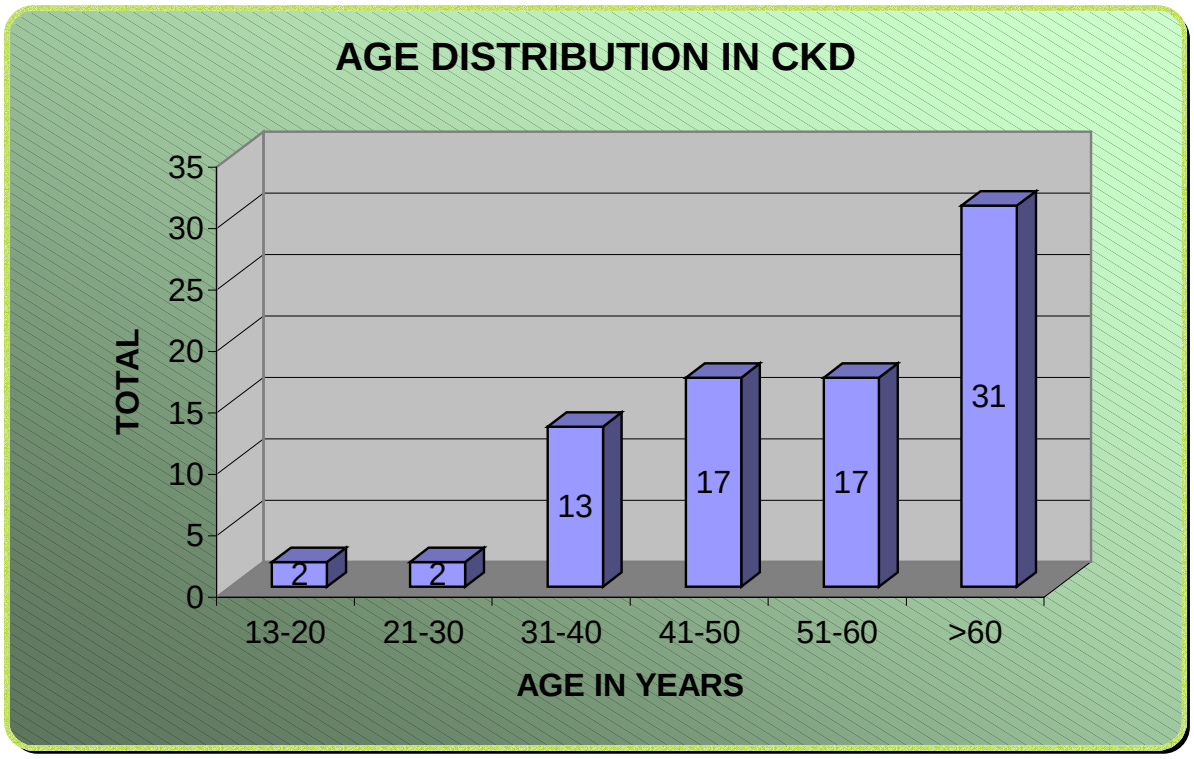

AGE DISTRIBUTION IN CKD
TOTAL
35
30
25
20
15
10
5
0
2
2
13
17
17
31
13-20
21-30
31-40
41-50
51-60
>60
AGE IN YEARS

FIGURE 6

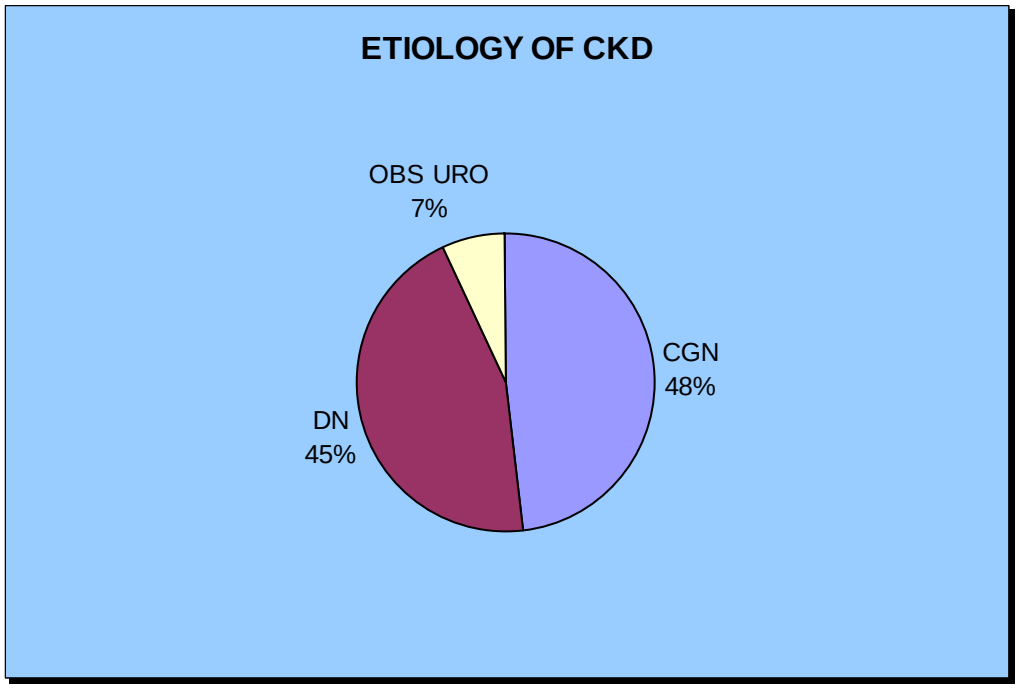
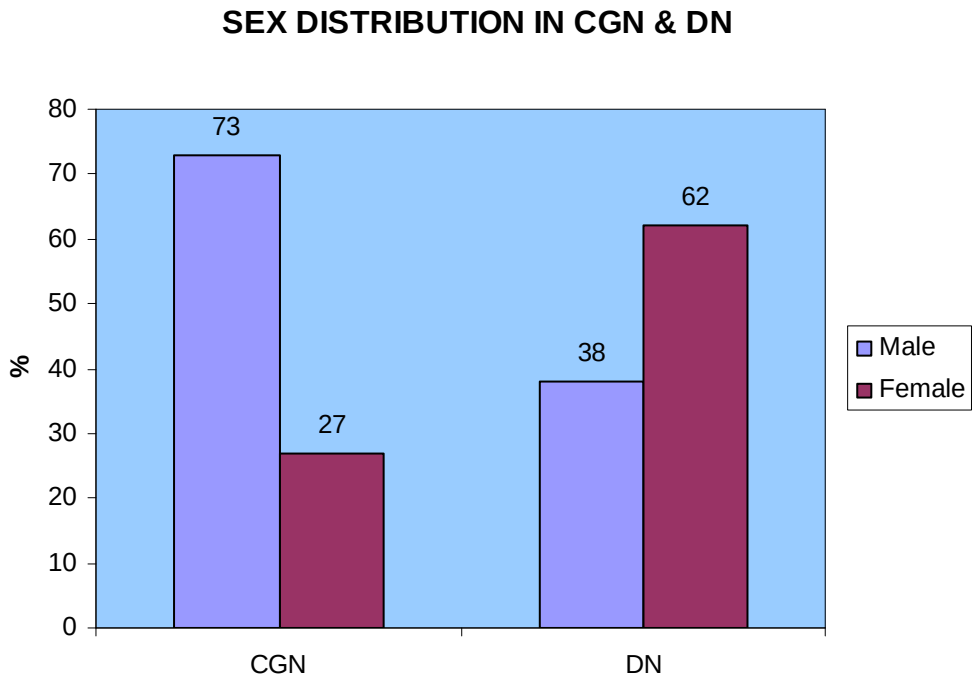


FIGURE 7



CLINICAL FEATURES OF CKD

FIGURE 8

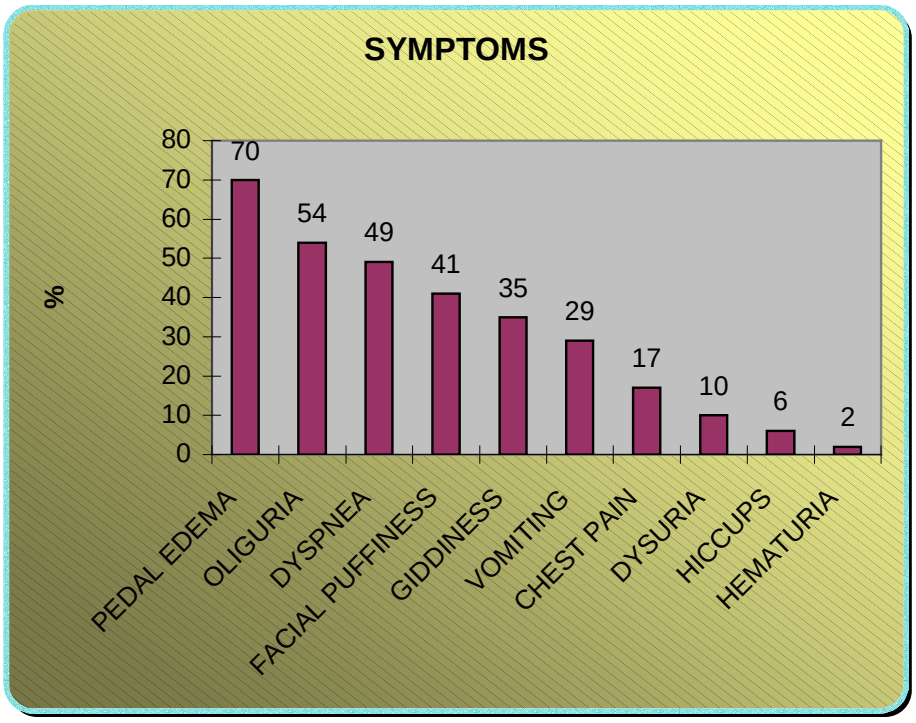


FIGURE 9